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THE OBESITY, MENSTRUAL IRREGULARITY AND HIRSUTISM SYNDROME

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Klerksdorp *

Until the pathogenesis of this endocrinopathy is understood and defined, the designation of this fairly common condition as the *obesity, menstrual irregularity and hirsutism syndrome* seems sufficient to fulfil the interim need. The syndrome usually manifests itself in young women with the sudden onset of obesity followed by menstrual anomalies and not infrequently accompanied by the development of slight hirsutism. Various patterns of development are encountered in this syndrome, the most typical being the post-marital type and the post-pregnancy variety.

1. *Post-marital Type.* The history usually obtained in this type of case is that of a young woman who has a normal puberty and menstrual pattern until marriage, and develops this condition soon afterwards. These patients develop a voracious appetite almost immediately after marriage and obesity follows during the next few months. The weight may increase by 30-100 lb. Soon they start experiencing menstrual anomalies, most frequent in the form of oligomenorrhoea. Not infrequently they notice the concomitant development of slight hirsutism. Furthermore, they often complain of an inability to become pregnant.

2. *Post-pregnancy Type.* A similar train of events not infrequently follows pregnancy, usually full-term but in some cases an abortion. Soon after the birth of the baby, and often also during pregnancy, the woman allows her weight to increase tremendously. Often lactation is unsatisfactory. During the ensuing months her periods become abnormal. The obesity increases and the oligomenorrhoea persists. In due course she finds she is unable to become pregnant again. It is then, or even earlier, that she is given various hormone preparations, whereas in actual fact the whole trouble can often be cured by plain and simple reduction of weight.

3. *Other Types.* Although the post-marital and post-pregnancy types are the most typical, various other patterns of development are also found. Often the history is obtained that the patient underwent an operation (not necessarily of any special nature) and after this she develops obesity with its consequences (*post-operative type*). At other times the history of a severe emotional upset is obtained, followed by a similar train of events (*post-stress type*).

SYMPTOMATOLOGY

Obesity is a classical component of the syndrome. It seems to affect the trunk and proximal parts of the limbs more than the distal parts.

Oligomenorrhoea is the usual menstrual pattern, i.e. infrequent menses occurring at intervals of less than 3 months. Often these intervals are more than 3 months; that is to say, periods of *secondary amenorrhoea* occur. On occasion, even with the history of oligomenorrhoea, the periods are clinically ovulatory in character and cramp-like pains are experienced on the first day of the flow. This, however, is exceptional. Usually the menses are completely painless and anovular. The occasional ovulatory character of these menses no doubt explains the rare occurrence of pregnancy when this syndrome has become established. Sometimes these infrequent periods are of a *metrorrhagic* type of bleeding. In rare cases regular menstruation is found. When this occurs it is more commonly anovular than ovular in nature.

Hirsutism, although frequently present, should not be regarded as a classical component of the syndrome. When present it is usually only slight in amount and takes the form of an abnormal growth of hair over the upper lip, chin, sternum and nipple regions, abdomen, lumbo-sacral region and limbs. Often the abdominal hair growth is attributed to shaving before an abdominal operation. It is revealing, if the point is enquired into, how frequently patients will state that the hirsutism started at about the

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same time as the obesity. Sometimes these patients volunteer that other members of their family are also hirsute. Whether patients with this familial trait are more inclined to develop this syndrome is not known.

Headache is another frequent complaint. Mostly it is situated over the vertex, but often it is typically migrainous in character. A family history of *diabetes* is not infrequently found.

DIFFERENTIAL DIAGNOSIS

Although the onset of hirsutism in any young woman may herald a serious disorder,[1,2] in the vast majority of cases the underlying endocrinopathy is not of such serious concern. It would be ideal if an estimation of the urinary 17-ketosteroids could be obtained in all hirsute patients in order to rule out those few cases where the basis of the disorder were a major *adrenocortical disturbance—neoplasia or hyperplasia*. This, however, is not practical except in institutions with facilities for endocrinological investigation, and a different approach to the problem has to be made. Should the history conform to that given above, there are for practical purposes only two conditions which merit consideration in the differential diagnosis, viz. the '*obesity, menstrual irregularity and hirsutism syndrome*' and *ovarian hyperthecosis*. The latter is not an uncommon condition. There is a great possibility that these two endocrinopathies are of an identical nature, being only different in the stage of development reached or in the emphasis on the various target glands. The only clinical point of distinction between the conditions is the fact that in ovarian hyperthecosis bilateral ovarian enlargement is present, whilst in the obesity, menstrual irregularity and hirsutism syndrome the ovaries are supposed to be normal. For this reason the importance of a careful vaginal examination must be emphasized. This may also lead to the discovery of the very rarely occurring *androgenic ovarian tumour*.

Since many of these patients are obese, and since the proper evaluation of the state of the internal pelvic organs is at times a very difficult, if not impossible, matter on ordinary vaginal examination, examination under relaxant anaesthesia is essential. Curettage (and possibly salpingography and pelvic aerography) done at the same time may aid in establishing the diagnosis. Should the ovaries be palpable as firm, enlarged, globular or elongated masses, most often lying in the pouch of Douglas (ovarian hyperthecosis), *bilateral subtotal ovariectomy* should be seriously considered; strict reduction of weight is then also essential. Should no ovarian enlargement be ascertained under these favourable conditions, *strict weight reduction* is of paramount importance. In many cases this will be all that will be required in the way of therapy.

So-called 'partial gynaecography'[3] has been extensively used in America in the investigation of these cases. In this procedure the uterine cavity is outlined by a radio-opaque substance, followed by the induction of a pneumoperitoneum to display the internal pelvic organs radiographically. Theoretically it should be of immense value, particularly in the obese patient, in view of the pitfalls of a vaginal examination, even if carried out under anaesthesia. However, the attempts at the procedure made at Groote Schuur Hospital have been disappointing.

PATHOGENESIS

The pathogenesis of this common and interesting condition is still obscure. The most reasonable explanation seems to be that it is a 'stress' reaction with a consequent upset in the hypothalamic-pituitary axis and resultant dysfunction of the adrenal cortex and ovaries. It seems to be most feasible that there is an adrenocortical dysfunction without neoplasia or hyperplasia. Jones and Jones[4] found increased quantities of pregnanetriol excreted in the urine in patients corresponding to these described types. Pregnanetriol is the urinary excretory metabolite of 17-hydroxyprogesterone, an androgenic substance found in the adrenal cortex during the synthesis of 17-hydroxycorticosterone.[5] Our own observations have shown that there is an increased excretion of so-called 'pregnanediol' in the urine of patients with ovarian hyperthecosis.[6] The method[7] used, however, might have resulted in pregnanediol being misinterpreted for pregnanetriol.[8,9] Another possible pointer to the probability of adrenocortical dysfunction in these cases is the finding that some of them respond to the administration of cortisone.[10,11]

Further work on this matter is at present being conducted at Groote Schuur Hospital.

SUMMARY

1. The 'obesity, menstrual-irregularity and hirsutism syndrome' is described.

2. The view is put forward that it develops as a consequence of a 'stress reaction' with resultant dysfunction of the adrenal cortex and ovaries.

3. The possibility that this endocrinopathy is basically the same as the condition of ovarian hyperthecosis, being only different in the stage of development reached or in the emphasis on the various target glands, is mentioned.

I gratefully acknowledge the interest and stimulus received from Prof. J. T. Louw during the years of my association with the Gynaecological Endocrine Clinic at Groote Schuur Hospital, Cape Town, and also the help received from my numerous colleagues who referred these cases for investigation. Mr. N. Constantine performed the technical investigations and to him also I am indebted.

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EDITORIAL

ADRENOCORTICAL HYPERFUNCTION IN CHILDHOOD

Hyperactivity of the adrenal cortex with bilateral hyperplasia may occur during foetal life. In this condition an excess of androgenic hormone is secreted, which produces a masculinization of the developing female embryo. Mullerian duct derivatives persist, so that the uterus and vagina (and of course ovaries) remain, but the clitoris enlarges and only a single perineal opening develops, representing a common vaginal and urethral aditus. This is the usual form of female pseudo-hermaphroditism, the congenital adreno-genital syndrome. The basic lesion (described in a previous editorial[1]) is a failure in the pathway of formation of glucocorticoids in the adrenal cortex. Androgenic steroids accumulate and the cortex hypertrophies, probably under the influence of an increased production of pituitary corticotrophin. If the condition remains untreated, a premature, male type of puberty occurs, with beard growth, further phallic enlargement, deepened voice, and male muscular development and contours. The adult appearance resembles that of a short man with hypospadias and cryptorchidism. Distinguishing features are a female nuclear sex-chromatin pattern, high 17-ketosteroid output in the urine, and the possession of a uterus and ovaries. Suppressive therapy with cortisone has been remarkably successful.

Hyperplasia of the adrenal cortex with excessive androgen production may start after birth. In this case the intrauterine sexual development has been normal, so that there is no intersexuality. In females, however, masculinization occurs, with a sort of premature male puberty, as mentioned above. The affected patients first show a growth spurt, but eventually end up short because of premature fusion of the epiphyses. In these cases, then, we get a person of male general appearance, but of female genital development with a much enlarged clitoris. This variety of the adrenogenital syndrome may be caused also by a benign adenoma of the cortex; in this case operation is necessary, but cortisone (or analogue) is again a most satisfactory treatment for the hyperplasia.

Hyperplasia or adenoma of the cortex in little boys does not of course produce any heterosexual affliction, but causes

VAN DIE REDAKSIE

OORPRODUKSIE IN DIE BYNIERSKORS BY KINDERS

Oormatige werking van die bynierskors, met weersydige oormatige groei, kan reeds gedurende die baarmoederlike lewe van die vrug voorkom. By hierdie toestand word 'n oormaat van die androgeniese hormoon afgeskei, waardeur die ontwikkelende vroulike vrug vermanlik word. Reste van die oergeslagsgang bly voortbestaan, sodat die baarmoeder en skede (en natuurlik ook die eierstokke) gevorm word, maar die clitoris word vergroot en slegs één uitmonding ontwikkel in die perineum—'n gemeenskaplike skede- en uretra-uitgang. Hierdie afwyking is die gewone vorm van die vroulike skyn-dubbelslagtigheid—die aangebore bynier-geslagsindroom. Die grondoorsaak of letsel (wat reeds in hierdie rubriek beskryf is[1]) ontstaan wanneer die glukokortikoïede nie in die bynierskors gevorm word nie. Androgeniese steroïede hoop op en die skors verskrompel, waarskynlik onder die invloed van 'n vermeerderde produksie van pituitêre kortikotrofien. As die afwyking nie behandel word nie, ontstaan daar 'n vroegtydige, skynmanlike puberteit met baard-ontwikkeling, verdere vergroting van die penis, 'n diep stem, en manlike spiere en voorkoms. Die volwasse voorkoms is dié van 'n kort man met hipospadie en kriptorchidisme. Kenmerkend hiervan is die vroulike kernpatroon van die geslagschromatien; 'n groot uitskeiding van 17-ketosteroïede in die urine, en die aanwesigheid van baarmoeder en eierstokke. Kortisoon is reeds met besondere welslae as onderdrukkende behandeling gebruik.

Oorontwikkeling van die bynierskors met oormatige produksie van androgeen kan kort na die geboorte ontstaan. In hierdie geval was die baarmoederlike ontwikkeling van die vrug normaal, sodat daar geen dubbelslagtigheid is nie. By dogtertjies is daar egter, soos reeds beskrywe, 'n mate van vermanliking met 'n soort vroeë manlike puberteit. Die pasiënte toon eers 'n fase van vinnige groei, maar die volwassene is nog kort van postuur omdat die beenente vroegtydig sluit. In sulke gevalle sien ons dus 'n persoon met 'n algemeen manlike voorkoms, maar met 'n vroulike geslagstelsel en baie vergrote clitoris. Hierdie voorbeeld van die bynier-geslagsindroom kan ook deur 'n goedaardige gewas in die bynierskors veroorsaak word; by hierdie gevalle is snykundige ingreep nodig, maar kortisoon (of 'n ooreenkomstige middel) is ook hier 'n baie bevredigende behandeling vir die oormatige weefselgroei.

Oormatige weefselgroei of 'n gewas in die skors by seuntjies veroorsaak natuurlik nie 'n vervrouliking nie, maar

an isosexual precocious puberty. This pubertal development is in all ways like that at the normal age except that, since it is caused by adrenal androgen and not by pituitary gonadotrophin, the testes may remain small and fertility does not develop. There is no spermatogenesis. This distinguishes the condition from the precocious puberty of pineal tumour or the 'idiopathic' variety. Again, the affected people end up short because of early epiphyseal closure; this type of adrenogenital syndrome produces the 'pocket Hercules'.

Over-production of glucocorticoids, leading to Cushing's syndrome in childhood, is quite a different kettle of fish.[2] Until very recently there was apparently no report of Cushing's syndrome before the age of 10 which was not caused by an adrenal carcinoma. (One case of bilateral hyperplasia and one of adrenal adenoma have been described within the last few years.) Now, whereas an adenoma is biochemically active in the production of only a single hormone, so producing the pure picture of Cushing's syndrome (in adults) or the adreno-genital syndrome, a carcinoma really goes to town and produces greatly increased quantities of androgen, glucocorticoid and oestrogens (probably aldosterone also). Consequently a mixed clinical picture is produced, usually with one or other hormone predominating. The little girl may then become very fat, with a podgy, cushingoid, plethoric face, downy hairiness, muscular weakness, cessation of growth, and glycosuria. The purple striae characteristic of Cushing's syndrome in the adult are not so commonly seen, while the obesity is frequently distributed over the lower extremities and arms as well as the trunk. In addition, there may be features of virilism, with growth of the clitoris and increase of muscle mass instead of muscle atrophy, and with growth of darker hair of male type on the face and abdomen and sexual hair at axillae and pubis. Bone growth and bone age are static if the Cushing's syndrome prevails; on the contrary they are advanced if the adreno-genital side is foremost. The majority of cases are in girls (as in adults, most Cushing's syndrome occurs in females)—in little boys the tumour tends to produce a mixture of Cushing's syndrome and isosexual precocity. Very rarely the over-production of oestrogens prevails, and feminization develops (in boys) or isosexual precocity (in girls) with breast development and premature menstruation. No case has yet been reported in which the major hormone produced has mineralo-corticoid effects.

The prognosis of these children with Cushing's syndrome is poor, but not uniformly so—in several cases complete cure has followed operative removal. In others the progress of the disease and the secondary spread of the neoplasm are so rapid that some of the classical features of chronic Cushing's syndrome, such as osteoporosis, do not have time to occur.

1. Editorial (1955): S. Afr. Med. J., **29,** 512.
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dit veroorsaak wel voorbarige manlike puberteit. Hierdie (vroegtydige) puberteitsontwikkeling is presies soos dié wat op die normale ouderdom voorkom, behalwe dat die testes miskien klein bly en die seun onvrugbaar is omdat die puberteit hier deur die skors-androgeen veroorsaak word en nie deur die pituitêre gonadrotrofien nie. Saadvorming ontbreek. Dit is hierdie kenmerk wat hierdie bepaalde afwyking onderskei van die vroegtydige puberteit wat veroorsaak word deur 'n gewas in die corpus pineale, en van die ‚idiopatiese' soort. Ook by hierdie gevalle bly die seuns kort van postuur omdat die beenente vroeg sluit; hierdie soort bynier-geslagsindroom veroorsaak die soort postuur wat as ‚pocket Hercules' beskryf word.

Oorproduksie van die glukokortikoïede, met die Cushing-sindroom by kinders as gevolg, is heeltemal iets anders.[2] Tot onlangs was daar skynbaar geen verslae van die Cushing-sindroom by kinders onder tien jaar nie wat *nie* deur 'n bynierkarsinoom veroorsaak is nie. (Één geval van weersydige oormatige weefselgroei, en één geval van 'n bynier-gewas, is in die afgelope paar jaar beskryf.) Maar 'n kliergewas is biochemies aktief by die produksie van slegs 'n enkele hormoon en lei dus tot 'n ongekompliseerde Cushing-sindroom (by volwassenes) of tot die bynier-geslagsindroom, terwyl 'n karsinoom aan die ander kant hoogs vermeerderde hoeveelhede androgeen, glukokortikoïede, estrogene (en waarskynlik ook aldosteroon) voortbring. Die gevolg is 'n veelsydige kliniese siektebeeld waarby een van hierdie hormone gewoonlik die ander oorskadu. Die dogterjie kan dan baie vet word, met 'n poffige Cushing-agtige volbloedige gesig, donsige harigheid, 'n swak spierstelsel, beperkte groei, en glikosurie. Die pers strepe wat by volwassenes so kenmerkend van die Cushing-sindroom is, word minder dikwels by die kinders gesien, en die vetsug strek dikwels oor die arms en bene sowel as die lyf self. Daar kan ook trekke van mansagtigheid wees, met oorontwikkeling van die clitoris en sterker spierontwikkeling in plaas van spieruittering, en die groei van donker mansagtige hare op die gesig, buik, en skaamhare by oksel en pubis. Indien die Cushing-sindroom die beeld oorheers, bly die beengroei en -ouderdom beperk; hulle loop egter vooruit as die bynier-geslagsindroom die bo-toon voer. Die meeste gevalle kom by meisies voor (by volwassenes word die Cushing-sindroom ook oorheersend by vrouens aangetref); by seuntjies is die gewas geneig om 'n mengsel van die Cushing-beeld en 'n eie-geslagtelike vroegrypheid te veroorsaak. Dit is baie selde dat oorproduksie van die estrogene die siektebeeld oorheers, en dan kom daar vervrouliking by seuntjies voor; die meisies bereik baie vroeg 'n vroulike puberteit met ontwikkeling van die borste en maandstonde. Dusver is daar nog geen verslag van 'n geval waar die oorheersende hormoon mineraal-kortikoïede gevolge gehad het nie.

Die prognose vir hierdie kinders met die Cushing-siektebeeld is maar swak, maar daar is uitsonderings—by 'n hele paar gevalle het die pasiënte volkome herstel na snykundige verwydering. By ander vererger die siekte só gou, en versprei die gewas sekondêr só vinnig, dat sommige van die tipiese aspekte van die Cushing-sindroom, soos beenverweking, glad nie kans het om te ontwikkel nie.

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THE TUBERCULIN REACTION

Many features of the mechanism of delayed hypersensitivity reactions, as exemplified by the tuberculin test, require investigation. It has been demonstrated in recent years that tuberculin is toxic to cells of hypersensitive animals (when applied to them in tissue culture), and that tuberculin hypersensitivity can be transferred by inocula of cells, especially the lymphoid cells, of the reticulo-endothelial system.

The influence of various specific and non-specific factors on the tuberculin reaction has recently been reviewed by Pepys,[1] who has made an attempt to demonstrate a pattern of the events leading to combination of antigen and antibody, and the development of the inflammatory response. He has shown that the character and intensity of the tuberculin reaction can be influenced experimentally by various modifications of technique. By the use of agents which modify the local cutaneous circulation and the permeability of capillaries and venules in the dermis the persistence of the tuberculin at the injection site in the skin can be considerably influenced. In order that a reaction should appear the antigen tuberculin must persist locally in adequate concentration and for a sufficient time. The local persistence of tuberculin is influenced by lymphatic absorption. By injecting histamine or adrenaline locally it was demonstrated that the reaction is modified. The effects were observed readily by using fluorescein as an indicator. Investigation also demonstrated that 'fixation' of tuberculin commences within a few minutes after injection. The rate and degree of 'fixation' has an important bearing on the mechanism of the test, and further work is needed to assess the nature and firmness of the 'fixation'. The relationship of immediate and delayed types of hypersensitivity may reveal the nature of the early 'fixation' of tuberculin.

Antibody as well as antigen is subject to modifications of a non-specific and specific nature. The specific reacting antibody has been studied, and much assistance has been gained from the demonstration of reticulo-endothelial tissue (the macrophages and especially the lymphoid cells) as a source of antibody. It has been shown that decreased or inhibited tuberculin reactions can be produced by reducing lymphoid tissue and cells, as by X-irradiation or specifically by the intravenous injection of tuberculoprotein. Studies with mononuclear cells have shown that their numbers and availability influence the extent of the reaction to tuberculin, and it would appear that the delayed nature of the tuberculin reaction may be due to the time required for a sufficient number of antibody-carrying cells to appear at the test site, where after reaction with the tuberculin an inflammatory response is excited. There appears to be an intimate relationship between the reacting antibody and the intact lymphoid cells. However, it still remains to be demonstrated whether preformed antibody can produce the tuberculin reaction without being taken up by specific lymphoid cells.

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MAMMARY TUBERCULOSIS PRESENTING AS CARCINOMA

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A series of 5 cases of mammary tuberculosis is presented. These patients were admitted to the Surgical Unit at Groote Schuur Hospital between the years 1951 and 1955.

CASE REPORTS

Case 1

F.C., a married Coloured woman aged 44 years, complained of a swelling in the right breast and right axilla for 1 month. There was no pain in the breast and no increase in the size of the lump during this period. No discharge issued from the nipple. She was otherwise well with no symptoms referable to other systems.

On examination, the patient appeared to be well. The right breast was enlarged, with extensive *peau d'orange*. The skin was slightly red and warm, but no defined mass was felt. The right axillary glands were enlarged, firm and fixed. One enlarged mobile gland was palpable in the right supraclavicular fossa and one in the left axilla. The other systems were normal. X-ray of the chest appeared normal.

A diagnosis of inoperable carcinoma was made and the patient was given a course of X-ray therapy to the breast (4,000 r). Ten months later the breast was considered normal by the patient, but some thickening and fibrosis was felt on palpation. The right axillary glands had disappeared. The gland in the left axilla was much larger than previously, and was removed. X-ray of the chest was repeated, and appeared normal.

Pathological Report: 'The specimen consists of numerous lymph nodes, the largest 6×5 cm. The appearances are those of caseous tuberculosis. Histologically, this is confirmed as an active spreading caseous tuberculosis which has involved adipose tissue beyond the node capsule.' (Fig. 1).

The wound healed well. A short course of anti-tuberculous chemotherapy was given. Five years later the patient was well, with no recurrence. Apart from some thickening of tissues in the axilla and axillary tail, the right breast was normal.

Case 2

A.T., a married Coloured woman aged 50 years. Two months previously the patient complained of a cough productive of mucoid sputum, and pleuritic pain in the left chest. She was given an injection by her doctor and these symptoms rapidly disappeared. About 3 weeks later a swelling appeared in the region of the left axilla, which rapidly increased in size and broke down, discharging a creamy material.

On examination, the patient looked well. In the upper and outer quadrant of the left breast were 2 small scars, between which was a firm rounded mass attached to skin and underlying muscle. Numerous firm mobile glands were present in the left axilla. X-ray of the chest appeared normal. The diagnosis lay between tuberculous adenitis and carcinoma of the axillary tail. The mass in the breast was excised, and was found to consist of caseous material with much surrounding fibrosis. The remaining glands were then excised.

Pathological Report: 'The specimen consists of axillary tissue

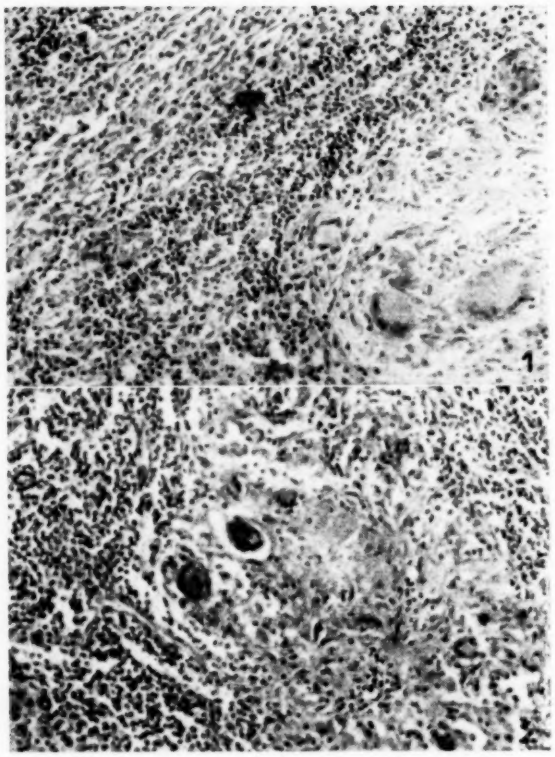

Fig. 1. Case 1. Typical tubercle follicle surrounded by tissue suggestive of fibrosed lymph gland.

Fig. 2. Case 2. Typical tubercle follicles in lymphoid tissue.

with overlying skin, weighing 220 g. There are many enlarged glands and fibrosis of the surrounding adipose tissue. The glands measure up to 2 cm. in diameter, and on section are white, containing small yellow foci. The histology is that of tuberculous lymphadenitis' (Fig. 2).

A short course of anti-tuberculous chemotherapy was given. The patient has not been traced since discharge from hospital.

Case 3

S.H., a married Coloured woman aged 50 years, complained of a painless swelling in the right breast for 1 month. During this period there was a noticeable increase in size.

On examination a hard, mobile mass was palpable in the right axillary tail and *peau d'orange* was present over more than half the breast. Firm, mobile axillary glands were present on the right side, extending almost to the apex. The other systems were normal. A diagnosis of inoperable carcinoma of the breast was made, and a full course of X-ray therapy was given (3,800 r to 4 fields). The mass in the breast and the glands became slightly smaller. A radical mastectomy was performed 2 weeks after completing treatment. Repeat X-ray of the chest appeared normal.

Pathological Report: 'The specimen consists of breast with underlying muscle and axillary fat. In the upper and outer quadrant 12·5 cm. from unfixed, non-retracted nipple, is a hard mass 3 cm. in diameter. On section the mass appears to be encapsulated, and section shows a partly translucent surface with opaque, pale yellow areas of ? caseation. In the axilla are 2 firm masses, 5·5×2·8 cm. and 4×1·8 cm. respectively, which on section have the same appearance. Histologically, the features are those of tuberculous adenitis. The inflammatory process is relatively inactive, although tubercle bacilli were demonstrated in material taken from the glands.' (Fig. 3).

A short course of anti-tuberculous chemotherapy was given. The wound healed well and there has been no recurrence 3½ years later.

Case 4

M.S., a married Coloured woman aged 67 years, was admitted with epistaxis and hypertension. A swelling in the right breast was discovered on routine examination. It had been present for 3 months, gradually increasing in size, and was painless. No discharge from the nipple had occurred.

On examination, a hard non-tender mass 1¾ inches in diameter was felt in the axillary tail of the right breast. It was attached to the underlying muscle but not to the chest wall. Fixed, hard axillary glands were present, including the apical glands. There was no oedema. The patient was hypertensive with no signs of cardiac failure. The other systems and an X-ray of the chest were normal. A diagnosis of inoperable carcinoma was made and a full course of X-ray therapy was given (3,800 r to 4 fields). Four months later the breast lump was slightly smaller and tender on palpation; the axillary glands were unchanged. A local mastectomy was performed.

Pathological Report: 'The specimen consists of—

(1) A breast with axillary tissue but no underlying muscle. In the upper and outer quadrant of breast tissue is an elongated mass of caseous tissue of 7 cm.×3 cm.;

(2) Two separate glands measuring 3 cm.×4 cm. in diameter respectively, both of which show extensive caseation. Histology is that of necrotic material. The fibrous encapsulation and occasional epithelial and giant cells would suggest a tuberculous aetiology despite the fact that tubercle bacilli are not demonstrable.' (Fig. 4).

The wound healed well, a short course of anti-tuberculous chemotherapy was given; 1½ years later there was no recurrence, but chronic oedema of the right arm of moderate degree was present.

Case 5

C.J., a married Coloured woman aged 36 years, complained of a swelling in the right breast for 2 years, gradually increasing in size, and painless. She first noticed it just before the birth of her last child. There was some recent loss of weight.

On examination, a swelling was felt in the right breast in the region of the axillary tail, 1×2 inches in size. It was hard, non-tender and mobile. Small, firm, mobile glands were palpable in the apex of the right axilla and in the right supraclavicular fossa. The other systems were normal, as was an X-ray of the

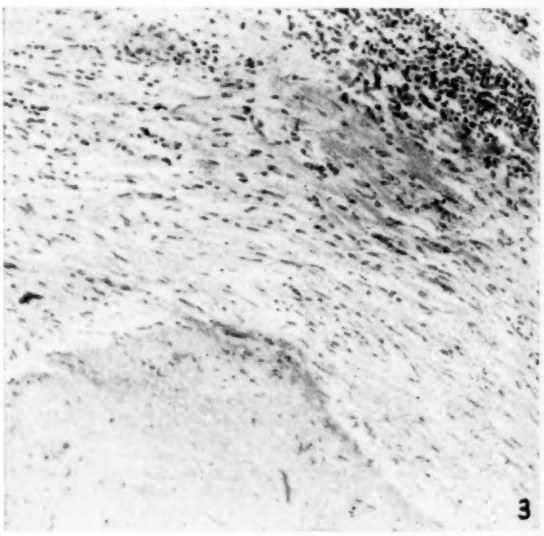

Fig. 3. Case 3. Caseous necrosis surrounded by dense fibrous tissue. Remnants of lymphoid tissue at periphery.

chest. A diagnosis of inoperable carcinoma was made and a full course of X-ray therapy given (3,800 r to 4 fields). The supraclavicular glands became smaller and the breast lump was recorded as 1×½ inches in diameter. The breast lump was excised, and when caseation was found, the axillary glands were also excised.

Pathological Report: 'The specimen consists of two pieces of adipose tissue, together weighing 24 g. In section, both reveal a similar appearance of multiple glands in which 90% of the

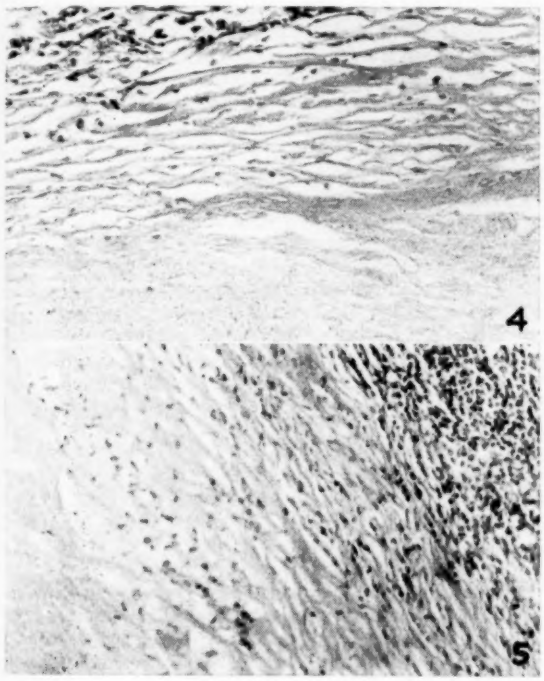

Fig. 4. Case 4. Caseous necrosis encapsulated by hyaline fibrous tissue. Remnants of lymphoid tissue at the periphery.
Fig. 5. Case 5. Encapsulated caseous necrosis in lymph node.

tissue shows coagulative necrosis. The histology is that of fibrocaseous tuberculosis. The necrotic tissue is surrounded by dense fibrous tissue, in which signs of active tuberculosis are slight. Acid-fast bacilli, though demonstrable, are scanty.' (Fig. 5).

The wound healed well, and 1½ years later the breast was normal, with no recurrence. A course of anti-tuberculous chemotherapy with INH and PAS was given for 7 months.

GENERAL DISCUSSION

Mammary tuberculosis was recognised in 1829 by Sir Astley Cooper as 'scrofulous swelling of the bosom'. Velpeau (1854) distinguished 3 forms of the disease, disseminated tuberculosis, lymphatic tumours, and lymphatic degeneration. Scott (1904) added a fourth variety which he called tuberculosclerosis, pointing out that the clinical features and macroscopic appearance was very difficult to differentiate from that of carcinoma. In 1903 Scott-Schley found that 65 cases had been recorded in the literature and in 1931 Morgan reviewed 439 cases.

Incidence. Mammary tuberculosis accounted for 1·5% of all patients with pathological conditions of the breast at St. Bartholomew's Hospital between 1890 and 1903 (Scott, 1904), and 1·7% of all such patients at the Memphis General Hospital between 1922 and 1934 (McGehee and Schmeisser, 1935). There was 1 case for every 200 cases of mammary carcinoma at the Mayo Clinic from 1909 to 1934 (Harrington, 1936). At Groote Schuur Hospital, 1-2 cases of mammary tuberculosis are seen each year, where the number of patients admitted with breast conditions averaged 146 per annum during 1954 and 1955. It occurs predominantly in married women, and a significant number occur during pregnancy or lactation.

Age. The disease occurs most commonly in the 3rd, 4th and 5th decade, with occasional cases in older people (Morgan 1931). In the 27 cases described by Scott (1904), the average age was 36 and the range 18 to 66 years.

Sex. Predominantly in females. Of Morgan's 439 cases, 22 were males.

Pathology. The lesion is usually one of two main varieties:

1. The nodulo-casious lesion, where caseation predominates and there is little fibrosis. Sinuses are prone to occur.

2. The fibrous type, where fibrosis predominates and there is little or no caseation.

Mode of Infection

The possibilities to be considered are: (1) Direct inoculation of the breast *via* the skin, with or without axillary gland enlargement (primary complex), (2) infection *via* the duct system, (3) Spread from the chest wall, (4) Haematogenous dissemination from a focus elsewhere, and (5) Lymphatic dissemination from a focus elsewhere.

The development of a primary complex involving breast and regional glands had not been reported in the literature, although tuberculous lesions have been reported in other parts of the body from a local primary inoculation. Inoculation *via* a wound in the breast leading to a local lesion only is no doubt possible, but infection through broken skin (as in a case quoted by Morgan (1931) of a woman with a breast lump which was massaged with ointment by a tuberculous husband with the subsequent discovery of both carcinoma and tuberculosis in the breast) is not convincing, and the ability of the tubercle bacillus to penetrate normal skin is doubted by Rich (1944). Duct lesions are equally rare; they have been periductal rather than intraductal, indicating that the pathogenesis of the lesion is less likely to be concerned with spread through the duct than from some other source, possibly the subareolar lymphatics.

If haematogenous dissemination were common, one would expect to find lesions of the breast in patients with miliary tuberculosis, but this does not occur. Nagashima (1925) investigated 34 patients dying from miliary tuberculosis and found no lesions on serial section of the breast; guinea-pig inoculation of breast tissue was negative. Raw (1924), with experience of 10,000 sanatorium cases, found 7 cases of mammary tuberculosis, of which 5 were due to spread from tuberculous glands in the neck, 4 extending to the axillary glands and by direct spread to the breast, and *via* the supraclavicular glands to the chest wall; 1 was due to direct spread from a tuberculous arthritis of the shoulder, and 1 was of doubtful tuberculous aetiology; none of these patients had pulmonary tuberculosis. Rich (1944), in discussing the apparent resistance of certain tissues to tuberculous infection, mentions heart, skeletal muscle, testis, ovary, pancreas, thyroid and parathyroid; the breast

may well be in the same catagory. The reasons for this selective resistance are not known.

Spread from the chest wall, i.e. from lesions in the ribs, costal cartilages, sternum or spine, is responsible for some cases. The vast majority of cases reported in the literature are clearly of lymphatic origin. The most important link between the breast and the thorax is the internal mammary chain of lymph nodes.

Route of Lymphatic Spread

The internal mammary chain of glands appears to be the principal group through which infection in the thorax can pass to the breast by retrograde lymphatic spread. Through the deep lymphatic plexus of the breast it connects with the pectoral glands, and also by a circuitous route through the cervical glands. There are connections with the internal mammary glands of the opposite side and, through these, with the opposite axilla. In addition, infected glands in the chain may break down and cause infection of the breast by direct extension through the soft tissues of the chest wall, or by infection of the overlying costo-chondral cartilages and thence to the breast.

The lungs are drained into the broncho-pulmonary glands, from which lymphatics pass through the superior and inferior tracheo-bronchial glands to the paratracheal glands. Afferents of these glands are joined by afferents from the internal

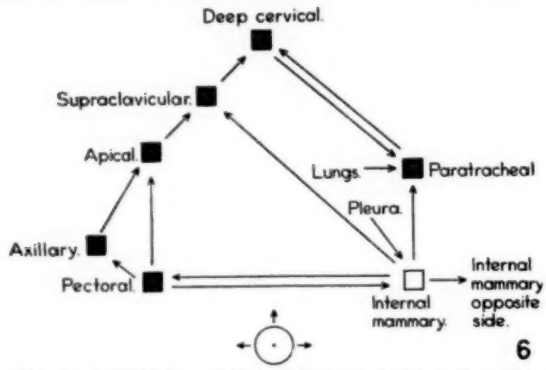

Fig. 6. Lymphatic connections between breast and mediastinum.

mammary glands to form the broncho-pulmonary mediastinal trunk. The greater part of the parietal pleura drains into the internal mammary chain. Infection may also pass from the cervical glands, when they are part of a primary complex, to the axillary glands, but this appears to be relatively uncommon (Fig. 6).

According to Burke (1954), the presence of tuberculous pleurisy, overt or occult, in mammary infection, may be of importance. He showed that radio-opaque material and dye injected into the pleural space of the guinea-pig is regularly conveyed to the internal mammary lymph nodes and to the retroperitoneal lymph nodes situated close to the descending aorta at the level of the lower thoracic and upper lumbar vertebrae. Intrapleural injection of tubercle bacilli had a similar effect; even when the dose was small and there was no macroscopic pleurisy these lymph nodes were enlarged, but only 2 of 23 guinea pigs showed enlarged tracheobronchial nodes. The results of autopsy examination of patients who died of pulmonary tuberculosis were comparable. A survey of 14 patients with cold abscess of the chest wall (parasternal), showed that all but 3 had an antecedent or co-existing tuberculous pleurisy. Burke noted that the bone overlying the nodes frequently showed degenerative change which would render it liable to infection.

In Morgan's series (1931) the swelling most commonly occurred in the upper and outer quadrant of the breast, and was insidious in onset and painless. The duration was commonly less than 6 months. The longest duration was 14 years, while in another case it was 8 years. In the lactating breast the course was more acute, the involved areas becoming red and tense and the patient complaining of pain. Of the 439 cases in the series, 15 also had carcinoma of the breast—a most important point, because it may be overlooked. He considered that the majority of his cases were of lymphatic origin. The majority had no signs of active tuberculosis elsewhere.

Raven (1949) reported a case in which the patient had a cold abscess presenting through the breast. The track was found to dip down into the mediastinum through an intercostal space close to the sternum. Forrest and Parkes (1955) reported a case in a woman aged 77, in whom the lesion presented in the upper and outer quadrant of the breast and was diagnosed as a carcinoma; it was tuberculosis of the pectoral lymph glands. The case came to autopsy (death of cardiac origin) and the tracheobronchial lymph nodes were found to be enlarged and caseating. The internal mammary nodes were not examined. The lungs showed old calcified or ossified foci. No other active tuberculous lesion was found.

The Lymphatic Lesion in the Adult and Elderly. Reinfection may, though rarely, occur in elderly people. A woman aged 69, who had nursed her tuberculous daughter, developed a tuberculous ulcer on the dorsum of the hand, followed by extensive fresh caseation of the axillary lymph glands (Pagel 1948). Late extension of a tuberculous process from a primary focus in the lungs is less frequent than from foci in the glands.

Endogenous Lymphoglandular Recrudescence

Acid-fast bacilli may remain viable in 'old' lymph nodes for many years, even when calcification is present (as shown by guinea-pig inoculation). Favourable circumstances such as diabetes, starvation, fatigue etc., may cause them to multiply. Such a process may be found at puberty, in early adolescence, and after the age of 45, unaccompanied by any progressive pulmonary lesion. It may eventually lead to progressive broncho-pulmonary tuberculosis, especially at puberty, or lymphatic spread to other glands may occur, or the focus may rupture into mediastinal structures, e.g. oesophagus, blood vessels or tracheobronchial tract. Ulceration of the stomach wall and melaena has been recorded (Pagel 1948). Drainage of the thoracic lymph trunks into the venous system may also lead to haematogenous dissemination, producing distant foci which may heal, or develop producing 'organ tuberculosis'. (Similarly, recrudescence may occur in a pulmonary primary focus.)

Endogenous lymphoglandular recrudescence in the mediastinal glands probably accounts for most cases of mammary tuberculosis, including those of this series with the possible exception of case 2, which have been due to recrudescence in a small sub-pleural lesion. The internal mammary group

of glands probably forms the link in the chain between the thorax and the breast, producing the breast lesion either by retrograde lymphatic spread to the pectoral glands or by direct spread through the chest wall. While it cannot be proved that haematogenous dissemination to the breast is rare, it seems likely that this is so, and certainly the well-documented cases in the literature suggest or show a connection with a caseating lymph gland.

DISCUSSION OF THE CASES

These 5 patients presented in a remarkably similar way with a swelling in the upper and outer quadrant of the breast. There was little or no pain. The duration varied from 1 month to 2 years, and the increase in size from a fairly rapid increase to little change. The final diagnosis in each case was tuberculous adenitis, involving the pectoral group of axillary lymph glands. The age incidence is a little higher than in most series. The occurrence in married women with children is usual. Pregnancy or lactation is not an uncommon association.

In case 2 the diagnosis of tuberculosis was suspected from the history of break-down of the lesion with sinus formation, but the residual mass could not be distinguished with certainty from a carcinoma; a tuberculous lesion does not rule out the possibility of a carcinoma in the same breast.

In the remaining 4 cases a confident diagnosis of carcinoma was made. This presents a problem; for the extensive glandular involvement and/or lymphoedema was a contra-indication to surgery in 4 cases, and the correct diagnosis was not made until after a full course of X-ray therapy had been given and palliative surgery undertaken. In case 5 the mass was examined before proceeding with mastectomy, because the similarity to case 4 was recognized, and local excision was then performed. The extensive X-ray therapy had no adverse effect in any case, and in case 1 it was followed by a remarkable remission. Admittedly, this case was not proved to have a tuberculous lesion of the right breast, but the subsequent behaviour of the left axillary gland, which was tuberculous, makes a presumptive diagnosis reasonable, and 5 years later the patient is well, with no sign of recurrence.

The differential diagnosis in patients presenting with sinus formation includes chronic pyogenic infection, actinomycosis, and possibly gumma, although this is now rare. Examination of the pus or a biopsy from the wall of the abscess may be required to establish the diagnosis. When the patient presents with a 'tumour', the differentiation from carcinoma is very difficult unless some sign such as redness or increased warmth of the skin or fluctuation is present to suggest inflammation. This may be present in the nodulo-caseous lesion, especially in lactating women, but is absent in the fibrous lesion. In the latter, the differential diagnosis includes traumatic fat necrosis, plasma-cell mastitis, carcinoma, and possibly the rare lesion sarcoidosis. It may even remain in doubt after histological examination. All the cases in this series are clearly of lymphatic origin, the pectoral group of glands being predominantly involved. None of the patients showed signs of active tuberculosis, but the history of a previous pulmonary episode with pleuritic pain in one patient may be significant (Burke 1954). The pathogenesis suggested is that of endogenous lymphoglandular recrudescence with retrograde lymphatic spread.

The racial incidence, all the patients being Cape Coloured, probably reflects the socio-economic circumstances of this group. The course run by tuberculosis is similar to that found in the European, and the incidence and course of carcinoma of the breast is also similar to that in the European. Had these patients been Africans, the diagnosis of tuberculosis would always have been seriously considered, because the African adult frequently reacts to tuberculosis as an unsensitized person, developing widespread glandular involvement, and comparatively seldom suffers from carcinoma of the breast.

After operation all the patients were given a short course of anti-tuberculous chemotherapy. The wounds healed by first intention and there was no recurrence.

Since surgery is usually required to establish the diagnosis, there have been few reports of treatment by chemotherapy alone. A case reported by McKeown and Wilkinson (1952) of a cold abscess in the breast, in which the only operation was a biopsy through healthy tissue, healed completely in 3 months after treatment for 6 weeks with PAS and streptomycin.

The problem of diagnosis, particularly in the patient with the fibrous type of lesion, remains unsolved. When the 'carcinoma' is considered operable there is no difficulty. An excision biopsy and examination of the mass is essential, and mastectomy can be avoided. Consideration of the diagnosis of tuberculosis is warranted by a lesion in the axillary tail of the breast with extensive lymph-gland involvement which appears from the history to have developed concurrently with the lump in the breast, and lymphoedema of the breast is also a common finding. Although a biopsy in inoperable carcinoma is usually considered undesirable, it is preferable to the alternative of a large dose of X-ray therapy, which may be associated with considerable morbidity; although radiotherapy has not proved harmful so far as the tuberculous lesion is concerned, and may even produce some degree of regression. Should the lesion prove to be a carcinoma, X-ray therapy may then be given immediately, followed by palliative surgery if necessary. The macroscopic appearance of the lesion in each of the cases reported was clearly that of tuberculosis.

Biopsy and conservative treatment may be sufficient, as in McKeown and Wilkinson's case, but excision followed by a short period of antibiotic cover is preferable, provided the lesion can be completely excised without undue hazard to major vessels and without deformity.

The appearance of calcification in the breast lesion or the axillary glands would be most helpful. It was absent in the cases reported, which is not surprising in an active and probably fairly recent lesion.

I wish to thank Dr. N. H. G. Cloete, Medical Superintendent, Groote Schuur Hospital, Cape Town, for permission to publish these cases. I am also grateful to Mr. G. Sacks and Mr. D. J. du Plessis for permission to report on their patients and to Prof. J. H. Louw for his encouragement. My thanks also go to Mr. G. McManus for the photographs.

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FACIAL PAIN*

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As the conditions which cause pain in the facial structures are many and varied, the subject is one of considerable complexity and, because it is not fully understood, even more controversy.

Almost all general practitioners will at some time be confronted with patients complaining of facial pain and in these times of specialization the condition also falls within the province of the ophthalmic and ear, nose and throat surgeons, the physician and general surgeon, the neurologist and neuro-surgeon, and also the orthodontist. As it is not practical to consider, in detail, all aspects of the condition, I propose to deal with it mainly from the point of view of the neuro-surgeon.

In order to introduce some clarity the following main headings are presented as a broad classification and I will develop the various sub-groups in my theme:

1. Local involvement of the facial structures.
2. The cranial nerve neuralgias.
3. Painful vascular disorders of the face.
4. Pain referred to the face from distant structures.
5. Miscellaneous neurological conditions associated with pain in the face.
6. Atypical facial neuralgia.

1. LOCAL INVOLVEMENT OF FACIAL STRUCTURES

Many of the facial structures when directly involved by disease processes present with pain as a major symptom. Conditions in this group include infections of the paranasal and mastoid sinuses, eye conditions, including glaucoma, dental conditions such as peri-apical abscess or an unerupted tooth, diseases of the ear, including otitis and petrositis, and other allied conditions.

It is apparent that treatment directed towards minor infections of the paranasal sinuses rarely alleviates severe facial pain. Infection of the frontal sinus may cause severe pain but the pain originating from the other paranasal sinuses is usually dull and chronic and, unless the physical signs, including radiograms, are unequivocal in demonstrating the presence of a major pathological condition, one would be advised to seek elsewhere for the cause of severe facial pain.

There is a rare condition associated with chronic suppurative disease of the parotid gland. This is Frey's auriculo-temporal syndrome, which is characterized by a burning pain on one side of the face, associated with flushing of the skin and sweating when the patient eats. Between attacks there may be a persistent hyperaesthesia so that the patient may complain of discomfort during shaving or similar stimulation of the skin. This syndrome is due to involvement of the auriculo-temporal nerve and is usually relieved by avulsion of that nerve.

Costen[1] drew attention to disease of the temporo-mandibular joint as a cause of facial pain, and it would be convenient to mention the syndrome at this stage so that the clinical features can be compared with those of other conditions which will be discussed in due course. Most of the patients presenting with this syndrome are over the age of 40 and are usually edentulous. The joint is subjected to excessive strain due to malocclusion or loss of molar support and overclosure of the jaw. It was thought that the symptoms were due to compression of the chorda tympani and auriculo-temporal nerves and pressure on the Eustachian canal, but Gaylor[2] has advanced the theory that they may be due to distortion of the thin tympanic plate, which ends in a free edge at the petro-tympanic fissure and which affects the 5th, 7th, 9th and 10th cranial nerves. The clinical features include ear symptoms and pain and irritative phenomena in other sites. There may be a continuous or intermittent impairment of hearing, with a feeling of stuffiness in the ear and a dull pain. Sometimes there are snapping noises associated with jaw movements, and tinnitus or vertigo may be present. Pain may also occur at the vertex or in the region of the occiput or mastoid process, and there is occasionally a burning sensation in the throat, the tongue and the side of the nose. Tenderness is present over the affected joint. The patient may complain of a dryness of the mouth, and herpes of the external ear or buccal mucosa may be present. X-ray studies of the joint usually show arthritic changes, erosion and abnormal mobility, and the pain will respond to an injection of local anaesthetic into the petro-tympanic fissure.[3] The treatment consists of increasing the vertical dimension of the jaw by various orthodontic procedures.

Malignant growths of the mouth, jaws or pharynx, especially those involving the posterior wall of the maxilliary antrum or the nasopharynx, may cause very severe pain, which can be successfully relieved by interruption of the trigeminal nerve pathways or by section of the nerve in the middle or posterior fossae. Even if the growth has apparently

* Being the subject matter of an address delivered to the Odontological Society of the Union of South Africa (Johannesburg Branch) on 13 March 1957.

been successfully treated by deep X-ray therapy, painful local radiation effects may follow and these will also respond to similar nerve section. If the malignant process affects the glosso-pharyngeal and second cervical nerves as well, then the posterior-fossa approach is indicated because of the accessibility of all three nerves at the same operative exposure. The presence of malignant disease, with its complications, does increase the mortality rate of these operations but it is nevertheless surprising how well elderly and cachectic patients tolerate these procedures.

Trauma to nerves as a result of fracture of the jaws or following dental extraction may result in continuous pain in the distribution of the affected nerve and here again interruption of the affected nerve pathways may be indicated for the relief of pain. It is to be noted that while peripheral nerve section may give temporary relief, the pain can be expected to recur within a few months as the result of nerve regeneration. Destruction of the ganglion cells with alcohol or pre-ganglionic section is not followed by nerve regeneration.

2. THE CRANIAL NERVE NEURALGIAS

(a) *Trigeminal Neuralgia*

Trigeminal neuralgia is a nervous disorder with characteristic clinical features. It is one of the few types of facial pain which, if correctly diagnosed and treated, can be cured with certainty, and it is for this reason that the condition should be recognized for what it is, so that prompt treatment can be instituted.

The symptoms consist essentially of a severe momentary paroxysmal pain strictly within the distribution of one or more divisions of the trigeminal nerve in the complete absence of any signs of impaired function of the nerve. It is to be noted that there is no spread of the pain across the midline.

The other cardinal features are that the patient is free from all pain (except occasionally for a mild residual burning pain) between the attacks and that the attacks can often be provoked by external stimuli, such as friction applied to the skin, the exposure of the face to cold water or to a draught, or by the movements of the jaws caused by speaking or eating. It often happens that these stimuli precipitate an attack only when applied to certain areas of the face—the so-called 'trigger zones'—the sites of which are commonly in the region of the naso-labial fold or the skin of the lower lip overlying the mental foramen. The pain may spread from the 2nd or 3rd divisions into the 1st, but it is rare for the 1st division to be primarily affected.

These painful attacks, which usually occur in persons over the age of 60, are best described as having a shooting or stabbing quality, and each spasm rarely lasts for longer than a few seconds or, at most, a minute or two. Initially the spasms occur only occasionally but with the passage of time the intervals become progressively shorter, so that eventually the unfortunate victim may be subjected to bursts of excruciating pain at frequent intervals during the day. When this occurs the patient is in a pitiable plight indeed, and in extreme cases he may refuse to speak and may even deny himself food so that malnutrition, with its attendant evils, may further rob him of his moral fibre to an extent where death itself would offer him his only relief.

The aetiology of trigeminal neuralgia has not yet been convincingly elucidated and, as a spontaneous cure is extremely rare, it is fortunate that interruption of the trigeminal pathways offers certain relief. The disease is not related to dental sepsis and the removal of teeth has no influence on its course. It must be admitted, however, that often in its earliest stages it may present as pain related to the teeth without any of the other characteristic features and it is only after dental extraction has been carried out that the true nature of the condition becomes manifest.

In the early stages, vitamin B12 or Trilene inhalations may be employed. These occasionally have beneficial effects, but because of the proxysmal nature of the disease it may well be that the apparent relief is in actual fact due to the occurrence of a spontaneous pain-free period. Dihydroergotamine has also been used but its evaluation is also subject to the same criticism.

Interruption of the nerve pathways is achieved by surgical section or by the injection of chemical blocking agents such as alcohol, phenol or boiling water[3] into the peripheral nerve or its ganglion. Interruption of the peripheral pathways is only temporary and the pain recurs when the nerves regenerate. Peripheral injection is sometimes used as a diagnostic measure in doubtful cases, or to see how the patient will react to the numbness of his face which follows interruption at the commonly indicated sites. It occasionally happens that the patient experiences unpleasant paraesthesias in the denervated skin (anaesthesia dolorosa) and may complain that these sensations are less tolerable than the original pain.

The most widely employed procedure in cases of trigeminal neuralgia is the surgical section of the sensory root proximal to the Gasserian ganglion through the middle-fossa approach. The approach is usually made extradurally, but in cases of excessive adherence of the dura to the floor of the middle fossa, possibly as a result of previous alcohol injection, the ganglion can be exposed intradurally. Section of the sensory root gives permanent relief and it is possible to preserve those fibres subserving the cornea and conjunctiva in cases where the pain is confined to the 2nd and 3rd divisions. The motor root, supplying the muscles of mastication on the affected side is easily identified and can be spared. The mortality rate of this operation is very low.

Alcohol injection of the ganglion requires considerable experience for its adequate performance and has the additional disadvantage that relief is not always permanent because of the difficulty in securing adequate destruction of the ganglion. Furthermore, it is not always possible to preserve corneal sensation, and a neuroparalytic keratitis may subsequently develop. This may, however, be offset by a prophylactic cervical sympathectomy. However, it is an important consideration that a ganglion block does avoid an operation and its employment is especially indicated in the aged or where all three divisions are involved. Surgical section is advisable when it is desirable and feasible to spare corneal sensation and in cases where ganglion block produces only short-lived relief, or where it has failed entirely.

Taarnhøj[4] has recently advocated decompression of the trigeminal nerve as it courses over the petrous ridge. In this procedure sensation of the face is undisturbed, but the operation has not been widely accepted and has not been carried out for a sufficiently long time to assess the permanency of its effects.

If it is suspected that a lesion in the posterior fossa may

be a possible cause of the trigeminal pain, the nerve can be exposed *via* the posterior fossa. The operation is not difficult but it does nevertheless carry a slightly higher mortality rate than the middle-fossa approach.

The descending root of the trigeminal nerve extends well into the medulla and may be severed in this region. Tactile sensation is preserved in the face and the danger of a neuroparalytic keratitis is reduced but the possibility of other side-effects and a higher mortality rate rather tends to offset the advantages. This operation is indicated in young persons, and in cases where the condition is bilateral and where one side of the face has already been rendered anaesthetic by a previous retrogasserian neurectomy.[5]

(*b*) *Glossopharyngeal Neuralgia*

The pain in this relatively rare condition is similar to that of trigeminal neuralgia but is situated in the region of the throat at the base of the tongue with radiation down the side of the neck, in front of the ear and to the back of the lower jaw. Exceptionally the pain may begin deep in the ear.

The attacks tend to be triggered by swallowing, coughing or protruding the tongue, and can be temporarily abolished by spraying the back of the tongue and pharynx with a surface anaesthetic. Malignant growths in the pharynx must be excluded. The paroxysms of pain can be terminated by sectioning the glossopharyngeal nerve in the posterior fossa but, if the ear is also involved, it is advisable to section the upper rootlets of the vagus nerve as well.[5]

(*c*) *Superior Laryngeal Neuralgia*

While pain in the region of the larynx is more often due to tuberculosis or cancer, a paroxysmal neuralgia may occur where no obvious pathological lesion is present. All these conditions respond to alcohol blocking or section of the superior laryngeal nerve. Harris[6] has injected alcohol into the internal laryngeal nerve. The paroxysms of pain are situated in the region of the thyro-hyoid membrane, with radiation to the side of the neck and up towards the lower face and gums and downwards towards the thorax and shoulder. Any irritation of the throat, such as swallowing or coughing, may trigger off the pain. Stimulation of the internal branch of the superior laryngeal nerve as it passes above the pyriform sinus or as it pierces the thyro-hyoid membrane may also provoke a paroxysm, and spraying the pharynx with a surface anaesthetic, or procaine blocking of the nerve, may produce temporary relief. Tenderness over the point where the nerve pierces the thyro-hyoid membrane may help in the diagnosis.[7]

(*d*) *Geniculate Neuralgia*

It was long thought that the facial nerve was purely motor in function but it has now been conclusively demonstrated that the nerve and its branches carry sensory fibres; the nervus intermedius is the sensory root and the geniculate ganglion is the sensory ganglion. Ramsey Hunt, during his observations on herpetic inflammations of the ganglion, has shown the cutaneous distribution to include parts of the pinna, the external auditory canal and the tympanic membrane.

Even in the absence of herpes, disturbances of the ganglion can lead to a severe deep lancinating pain in these regions, with radiation to the mastoid and retro-orbital and paranasal structures. Stimulation of parts of the ear may sometimes trigger off the pain and there may be a trigger zone in front of the ear.

The condition responds to section of the nervus intermedius if, at operation under local anaesthesia, the same pain is produced by stimulation of that nerve. If this association cannot be definitely demonstrated, it is advisable to section other nerves which supply the ear, namely the glossopharyngeal and the upper rootlets of the vagus.[8]

3. PAINFUL VASCULAR DISORDERS OF THE FACE

(*a*) *Facial Migrainous Neuralgia*

The facial pain in this condition has special characteristics with regard to the periodicity of its onset. The many cases of facial pain described under the names of Horton's histaminic headache, ciliary neuralgia, petrosal neuralgia, and Sluder's sphenopalatine neuralgia, suggest that these are merely synonyms of the same condition.

Facial migrainous neuralgia occurs predominantly in males, and consists of paroxysms of the sudden onset of a strictly unilateral, burning, constant pain in the supraorbital region or in or behind the eye and temple. Occasionally there is radiation to the face, jaw and neck. Each paroxysm of pain rises rapidly to a peak and then, after maintaining this level for from a few minutes to two or three hours, gradually subsides to disappear completely, leaving the patient free from pain. There is at least one attack in every 24 hours, the attacks being mainly nocturnal and often occuring with clock-like regularity, awakening the patient at the same hour each night.

This state of affairs may last up to 2 months, and is then followed by cessation of the pain with complete freedom for several months or even years before the attacks again reappear.

About half the patients have associated signs of autonomic disturbance in the way of congestion of the conjunctiva, swelling of the peri-orbital tissues, blockage of the nasal passage, and dilation of the temporal vessels.

The condition differs from migraine in that the attacks are of shorter duration and, although nausea may occur, there is rarely vomiting or visual or sensory disturbances. In addition, true migraine lacks the autonomic changes and the marked regularity of the attacks.

Treatment has been carried out in the past with varied results by histamine desensitization, by section of the greater superficial petrosal nerve[9] or by injection of the sphenopalatine ganglion. Sir Charles Symonds[10] has recently shown that the paroxysms can be prevented by the regular administration of intramuscular ergotamine tartrate until the patient enters his pain-free period, when the injections can be discontinued.

Dott[2] has stated that interruption of the trigeminal nerve gives certain relief.

(*b*) *Temporal Arteritis*

This disorder is part of a generalized vascular disease[11] occurring in elderly patients. A subacute inflammatory process involves the media of the arteries, and giant cells are present. The characteristic clinical features include fever and sweating, with anorexia and joint and muscle pains. Headache, which is severe and constant, is a major feature and visual disturbances are common. Temporo-mandibular pain occurs fairly frequently. When the superficial temporal

arteries are involved they are acutely tender and may become thrombosed, so that the pulsations are decreased. The disease tends to run a slow course and finally becomes arrested, leaving a variable degree of disability.

Relief may be obtained by the use of vasodilators or potassium iodide in large doses. Excision of small portions of the affected arteries may afford prolonged freedom from pain.

4. PAIN REFERRED TO THE FACE FROM DISTANT STRUCTURES

Diseases involving the heart and lungs may cause pain which radiates to the face, as may also pathological processes involving the cervical spine.

In cases of prolapsed intervertebral disc or cervical spondylosis the patient may present with orbital or peri-orbital pain with radiation to the temple or towards the vertex. There may be pain in the mastoid region or at the occiput.[12] The pain is constant, with variations in intensity, and is affected by emotional disturbances or by changes in the weather. Movements of the neck may influence the pain, and also any actions which raise the cerebrospinal-fluid pressure such as coughing or straining at stool.

Delmas, Laux and Guerrier, as quoted by Neurwith[12] have described the presence of sympathetic fibres in the ventral nerve roots C5, C6, C7, C8 and T1 and, as the pain often corresponds to the fields of arterial distribution of the external carotid system, it is supposed that the facial pain can be explained on a basis of sympathetic irritation. In any patient with facial pain of undetermined aetiology, and especially if there is evidence of cervical root involvement with motor and sensory signs, it is advisable to carry out radiological studies of the cervical spine and, if indicated, to institute treatment with traction or by means of a cervical collar.

5. MISCELLANEOUS NEUROLOGICAL CONDITIONS ASSOCIATED WITH FACIAL PAIN

Neurological conditions having facial pain as one of the symptoms are numerous, and include tabes dorsalis, disseminated sclerosis, intracranial aneurysms, posterior-inferior-cerebellar thrombosis and post-herpetic neuralgia. In these conditions the history and physical findings will give a clue as to the diagnosis, and treatment where indicated can be carried out.

In post-herpetic neuralgia the lesions probably involve both the peripheral and central nervous pathways and treatment directed towards the peripheral pathways alone is not likely to succeed. In severe cases where peripheral alcohol injections have failed one may have to resort to prefrontal leucotomy or other allied procedures, but it is always worth while to try the effect of deep X-ray therapy first.

6. ATYPICAL FACIAL NEURALGIA

It is unfortunate that, all too frequently, patients present with severe facial pain and yet the clinical features are not covered by those described in the foregoing groups. For want of a better term, these patients are said to be suffering from 'atypical facial neuralgia'.

It is considered by many that this type of facial pain is psychogenic in origin, but there are certain features which seem common to all these unfortunate sufferers and this tends to suggest that there is an organic basis for the pain. Admittedly there is a psychogenic overlay in many of the patients, but this is possibly merely the reaction to a demoralizing and debilitating condition. Probably the condition is due to irritative lesions involving the thalamus or thalamic connections in the brain or brain stem. Dott[2] believes them to be vascular in the older age-groups and neuro-degenerative in the younger.

The disturbance of sensation known as 'thalamic over-reaction', although rare in a fully developed form, is well recognized and is thought to be due to damage to the lateral nucleus of the thalamus. In this condition the pain is deeply situated and is constant and gnawing. Although the threshold for pain is raised, the stimulus, when effective, arouses sensations of a peculiarly unpleasant character and all sensory modalities evoke a disagreeable sensation. A particular stimulus applied to the affected side causes a more severe reaction than when it is applied to the unaffected side, and the precise localization of the stimulus is impaired.

In patients with atypical facial neuralgia these characteristics are present in a very modified form but do nevertheless serve as a basis for the pain usually described. These patients complain of a constant, ill-defined, deeply situated and poorly localized pain. They often describe it as being drawing or crushing or tearing. The pain is often bilateral and cannot be related to any particular nerve and there are no 'trigger zones'. These patients often over-react to their pain emotionally and drug addiction is common. Careful examination always fails to reveal any local or intracranial cause for the pain.

The treatment of atypical facial pain is always difficult and, more often than not, unsuccessful. Dott[2] has stated that on no account should any peripheral nerve injection or operation be done and many other neuro-surgeons, from bitter experience, also hold this view. Some cases do, however, respond to interruption of the peripheral pathways for a limited period until nerve regeneration takes place and in these cases a retrogasserian neurectomy may be indicated. Sympathectomy has been tried, with discouraging results. Psychotherapy may be of value and should always be tried before any surgical procedure is advised. If the patient has deteriorated to such an extent that he is already unable to hold his place in society, prefrontal leucotomy may be indicated, but here again the results are variable and relapses are frequent.

The question of Sluder's sphenopalatine neuralgia is always raised in a discussion of facial pain. Sluder described a pain of wide distribution starting at the root of the nose and extending on to the face and head and sometimes as far as the arm and finger-tips. The pain does not appear to have any specific characteristics, and it is difficult to know in any case presented as one of Sluder's syndrome that Sluder himself would agree with the diagnosis. For this reason the diagnosis of Sluder's neuralgia as a definite type of atypical facial pain is best avoided. Sluder has claimed successes in the treatment of atypical facial pain by cocainization or extirpation of the sphenopalatine ganglion, but this has not been the general experience. However, the injection of alcohol or hydrocortisone into the ganglion is still a common practice and this will no doubt continue until a more specific form of therapy has been advanced for this difficult condition.

SUMMARY

Facial pain is discussed under the following headings:

1. Local involvement of the facial structures
2. The cranial nerve neuralgias:
   (*a*) Trigeminal neuralgia
   (*b*) Glossopharyngeal neuralgia
   (*c*) Superior laryngeal neuralgia
   (*d*) Geniculate neuralgia
3. Painful vascular disorders of the face:
   (*a*) Facial migrainous neuralgia
   (*b*) Temporal arteritis
4. Pain referred to the face from distant structures with special regard to cervical pathology
5. Miscellaneous neurological conditions associated with pain in the face
6. Atypical facial neuralgia with a note on Sluder's syndrome.

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THE USE OF SYSTEMATIC DESENSITIZATION IN PSYCHOTHERAPY

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As the result of experiments conducted during the years 1947-48, Wolpe[1] was the first to systematize the principle of reciprocal inhibition in its application to the field of psychotherapy. He provides evidence that neurotic behaviour is 'persistent unadaptive learned behaviour in which anxiety is almost always prominent and which is acquired in anxiety-generating situations'.[2, 3] Successful therapy of the neuroses, therefore, would depend on the reciprocal inhibition of neurotic anxiety responses, i.e. the suppression of the anxiety responses as a consequence of the simultaneous evocation of other responses which are physiologically antagonistic to anxiety. Wolpe[4, 5] constructed an elaborate therapeutic system based on the assumption that if a response which is incompatible with anxiety can be made to occur in the presence of anxiety-producing stimuli it will weaken the bond between these stimuli and the anxiety responses. Whereas most psychotherapists report cured or improved cases in the vicinity of 60%, Wolpe claims a 90% level of cures or 'marked improvements' with his methods. By applying the x^2 test for significance, he proved that it is highly improbable that the higher proportion of his successes are due to chance factors. Arising from this, the following query has frequently been raised: Would Wolpe's techniques prove as effective in the hands of other therapists?

This paper illustrates that Wolpe's technique of 'systematic desensitization based on relaxation' has proved highly effective in the treatment of anxiety states by two independent psychotherapists. The illustrative cases were treated individually by one or other of the co-authors. Although the limited scope of this article covers primarily the use of desensitization, Wolpe has described a wide range of therapeutic methods to cover the entire field of neurotic behaviour disorders.

The rationale and application of systematic desensitization based on relaxation first appeared in this *Journal* in 1952. It involves a planned attack on neurotic anxieties, designed to reciprocally inhibit these unadaptive reactions by means of relaxation responses. Jacobson[6] has shown that intense muscle relaxation affects the autonomic nervous system so that the characteristic effects of anxiety are inexorably suppressed. It is therefore to be taken as axiomatic that relaxation inhibits anxiety—their concurrent expression is physiologically impossible.

PROCEDURE

Wolpe's articles[4, 5] on reciprocal inhibition therapy contain descriptions of numerous types of techniques. Because the present article is restricted mainly to systematic desensitization, we propose to present a detailed description of this procedure only.

An inquiry is first conducted in order to ascertain which stimulus situations provoke anxiety in the patient. The patient is told that he can add to or modify this list at any time. The stimuli are then categorized by the therapist and the patient is asked to rank the stimuli in order, from the most to the least disturbing. This ranked list of noxious stimulus conditions is referred to as the hierarchy. In case 3 for example, one would refer to the 'ambulance hierarchy' and the 'hospital hierarchy.' Hierarchies can contain from 5 to 25 items. The hospital hierarchy mentioned above consisted of the following stimulus situations: a hospital in the distance, a hospital ten corners away, walking past the hospital, standing outside the gates, walking in the grounds, standing outside the foyer, in the foyer, walking in the corridors, standing in a small ward of 4 beds, in a larger ward, in a surgical ward with a few bandaged people in bed. The construction of the relevant hierarchies generally takes 1-3 interviews and the patient is concurrently given practice in hypnotic and relaxation procedures. Hypnosis is not an essential requirement, and in those cases where the patient refuses to be hypnotized or requires prolonged practice the procedure can be omitted and deep non-hypnotic relaxation employed instead.

When the hierarchies have been worked out, the subject is told which stimuli are to be presented in the individual session and advised to signal with his hand if a stimulus presentation disturbs him unduly. This is an important instruction and should on no account be omitted, for the arousing of anxiety during the session is sometimes extremely damaging. In our experience it has been found that with most patients it is possible by closely observing his facial expressions, bodily tension, respiration and so forth, to perceive such disturbances before the patient actually signals. When such disturbances occur the therapist immediately 'withdraws' the stimulus and calms the patient. No session should be concluded when a disturbance occurs, but before rousing the patient the therapist should continue and present a further 'easy' stimulus which has already been successfully overcome. The reason for this is to be found in the commonly observed fact that the last item of any learning series is well retained.[7] Anxiety which occurs at the *end* of a session is likely to require a longer period before dissipating.

When the preliminary instructions have been given, the patient is relaxed (hypnotically or otherwise) and then told to visualize the various stimuli; e.g. 'Picture a hospital in the distance . . . Now stop picturing that and go on relaxing.' Each stimulus is visualized for 5-10 seconds and 2-4 different items are presented each session. Each item is generally presented twice. When the requisite number of stimuli have been presented the patient is slowly roused and then asked for a report on his reactions. If the items were visualized vividly and without undue disturbance, the therapist then proceeds to the next stimuli in the following session. The items lowest in the hierarchy (i.e. the least disturbing ones) are introduced first and the therapist proceeds slowly up the list depending on the progress achieved and the patient's reactions. In this way it is possible for the patient to eventually picture formerly noxious stimuli without any anxiety whatever. This ability to *imagine* the noxious stimulus with tranquillity then transfers to the real-life situation (see below).

ILLUSTRATIVE CASES

Case 1

A married woman of 34 was referred for treatment of an anxiety neurosis of 5 years' duration. She had received intermittent treatment during this period, including a brief spell of psychoanalysis, without apparent success. Two weeks before her first interview she had been advised to consider the possibility of undergoing a leucotomy.

She complained of attacks of fear with sweating, trembling and severe headaches. A wide variety of situations appeared to provoke these attacks, which tended to occur most severely and frequently in the late afternoon and in dull, overcast weather. The anxiety-producing situations included walking in the street, being outdoors in the afternoon, shopping, telephoning, crowds of people, and places of public amenity. She also reported an inability to cope in social situations and disturbing feelings of inadequacy and inferiority. Her sexual activity had been disrupted in recent months as the anxiety had increased, and was unsatisfactory. She had been taking 2-3 'tranquillizing' tablets per day for a short period with slight, variable results.

Application of the thematic apperception test and the Willoughby neurotic tendency inventory[8] revealed neurotic trends such as guilt, hypersensitivity and a marked lack of confidence (the Willoughby score was extremely high—87—indicating severe neurotic disturbance).

The patient was instructed in the use of assertive responses and deep (non-hypnotic) relaxation. The first anxiety hierarchy dealt with was that of dull weather. Starting from 'a bright sunny day' it was possible for the subject to visualize 'damp overcast weather' without anxiety after 21 desensitization sessions, and 10 days after the completion of this hierarchy, she was able to report that, 'The weather is much better, it doesn't even bother me to look at the weather when I wake up in the morning' (previously depressing). In addition to this improvement she was also able to go out for short periods during the afternoon. The following hierarchies were then dealt with: telephoning, shopping, having guests at the house, walking in the street, going to places of public entertainment, sitting in the garden in the afternoon.

Two weeks after the completion of the last hierarchy, the patient was given the Willoughby test again. Her score had dropped 40 points to the slightly inflated score of 47. There was also increased sexual responsiveness, a slight improvement in interpersonal relationships and increased self-confidence. The patient was now taking a refresher course in stenography with the intention of obtaining employment. She had not worked for 7 years. She voluntarily reduced her dose of 'tranquillizers' to one a day and dispensed with them completely 1 week later.

At this stage the patient's husband fell seriously ill and she was able to support him emotionally despite the considerable effort involved. As her husband's health improved, she suffered a minor relapse for 2 weeks and then returned to her improved state spontaneously. (A similar post-stress reaction has been reported by Basowitz *et al.*[9] in their study of paratroop-trainees in the US. These observations suggest an interesting and profitable line of investigation.)

During the course of therapy, part of the reason for the development of the anxiety state in this patient was unearthed. When she was 17 years old she had become involved in a love affair with a married man 12 years her senior. This affair had been conducted in an extremely discreet manner for 4 years, during which time she had suffered from recurrent guilt feelings and shame—so much so, that on one occasion she had attempted suicide by throwing herself into a river. It was her custom to meet her lover after work *in the late afternoon*. The dull weather can be accounted for, as this affair took place in London.

After 8 months of treatment, comprising 65 interviews devoted largely to systematic desensitization, this patient was 'much improved' in terms of Knight's 5 criteria.*

Case 2

A 32-year-old medical practitioner stated that he had developed a condition of 'psychic impotence'. He emphasized that he was already fully aware of the aetiological considerations—he first experienced sexual difficulties 3 months previously when he was harassed and in a state of tension. 'Since then, I enter sex with a feeling of uncertainty and am frequently unsuccessful.'

This case is atypical in that no 'anxiety hierarchy' was constructed, but the patient was conditioned to become completely relaxed before the sex act. This required 8 treatments in all. After 4 interviews he had become proficient at relaxation and systematic desensitization was then begun. This consisted of his visualizing certain pre-coital scenes accompanied by hypnotic relaxation. In a 17-month follow-up there has been no recurrence of the disturbance.

Case 3

A 14 year-old boy was referred for treatment of a 'simple' phobia. He had suffered from a fear of ambulances and hospitals for a period of 4 years. He stated that he was frightened by the sight of ambulances and avoided them wherever and however possible, e.g. by planning his journeys in advance and changing direction when an ambulance was sighted. He reported having fainted on several occasions when an ambulance was near by. He was also scared of hospitals and nursing homes and refused to visit these institutions.

His social and scholastic adjustments were both satisfactory and systematic desensitization was commenced after an initial period of training in relaxation. Separate hierarchies of noxious situations were constructed for the ambulance and hospital phobias. The ambulance-hierarchy ranged from easy (non-disturbing) stimuli such as a parked ambulance in the distance and a derelict ambulance in a scrap-yard, to difficult ones like sitting in an ambulance (*a*) next to the driver or (*b*) in the back. In the hospital-hierarchy the first easy situation was a distant hospital which could be barely seen and the final one, a surgical

* Symptom improvement, increased productivity, improved adjustment and pleasure in sex, improved interpersonal relationships, increased stress-tolerance.[10]

ward. Three days after the third desensitization session, the subject walked past a parked ambulance with its rear doors open and experienced no anxiety. Two further situations of a similar nature occurred during the course of therapy and neither of these evoked fear. After 10 interviews he was much improved and was able to visit the hospital and approach ambulances without difficulty. After a 3-month period there has been no recurrence of the earlier fears. A prolonged follow-up of this case is being undertaken.

Case 4

A 34-year-old engineer was treated for a speech disturbance characterized by lengthy and frequent 'word blocks', accompanied by considerable tension and facial grimaces. When first interviewed he stuttered on about 12-25% of words, with 'blocks' averaging 3-4 seconds. His attitude towards speaking situations was poor and he experienced difficulty in handling inter-personal relationships. His Willoughby score was 57, indicating a high basal level of neuroticism. He received 30 hours of therapy over 9 months. Therapy sessions were usually administered once a week. Training in progressive relaxation was followed by systematic desensitization. Among others, the following hierarchies were treated: time-pressures (especially speaking on the telephone, as he conducted many of his occupational affairs by long-distance calls), telling jokes, public speaking, difficult 'audiences' i.e. specific people with whom he had speech difficulties.

Progress was gradual, but by the termination of therapy a substantial gain in speech fluency had been achieved. He is still seen once every 3 months and the gain appears to be permanent, with occasional deteriorations occurring during periods of stress. He is under instruction to continue relaxation and solitary speech practice at his own convenience, and also to increase the frequency and duration of these activities whenever a period of stress is encountered.

Two innovations in this case should be mentioned. The patient was instructed to practise daily speaking (reading) aloud when alone. The rationale for this procedure is based on the 'spread of effect' phenomenon, positive transfer of training, and the observation that most stutterers speak more fluently when not in company. Stuttering appears to be essentially a *social phenomenon*. The second modification employed in the present case was that of speech practice in noxious situations (e.g. a public hall) under controlled conditions of relaxation. The patient was required to make a public address and accordingly was made to practise the actual speech in the hall on 6 occasions before the actual event, alternately relaxing and speaking. In this way he was enabled to make the speech with only a few blocks when the event took place. Generally, the symptoms were all greatly reduced and the stuttering pattern characterized by very occasional complicating sounds or facial grimaces. His Willoughby score had decreased. During the period of therapy he had become engaged and seemed to be managing inter-personal relationships more easily. He was recently married and delivered a completely fluent wedding speech.

Case 5

A married woman of 29, who had been a competent theatre sister in England for a number of years. She stated that from as far back as she could remember she had been a tense and anxious person, but as the result of a traumatic incident at the age of 24 she developed overwhelming phobic reactions to dogs. Accordingly, she underwent more than 3 years of psychoanalysis, but towards the end of this period her condition had deteriorated and, as it was interfering with her work, she was forced to resign from the hospital. In the hope that a change of environment might improve her condition, she and her husband came to South Africa. When first interviewed, she said that while she had overcome her fear of dogs, she was in a state of chronic anxiety and felt that suicide was her only release. Her rigid posture with perpetual tremblings and clammy hands indicated a deep-seated anxiety condition. It seems that the psychoanalytic treatment had merely blanketed her specific phobia with general anxiety.

Treatment consisted, in the first place, of 15 training periods in progressive relaxation. After 6 weeks' treatment (28 interviews) she responded well to hypnotic techniques and a further 4 interviews were devoted to deep hypnotic relaxation. At this stage the patient reported that she was generally relaxed and complacent in nearly all situations but that her original phobia for dogs had returned. An anxiety hierarchy was constructed and systematic desensitization applied. At first she reported acute disturbance when visualizing the feeblest scene (a small dog in the distance) but after 25 hypnotic desensitization treatments she was able to report only relatively mild anxiety when visualizing herself near a group of angry and snarling dogs. It took a further 4-5 weeks, however, before the patient was able to enter real-life situations involving dogs without experiencing acute anxiety (Wolpe states that there is sometimes a tendency for the real-life improvement to lag behind somewhat). At this stage, the patient was discharged from therapy, but returned about 3 weeks later and complained that her original phobia for dogs had returned. After only 3 further hypnotic desensitization treatments her phobia disappeared. She was seen 7 months later before returning to England. She reported that she was perfectly well and that she was returning to her previous hospital job; 5 months later she wrote a letter stating that she was still completely over her trouble and had not relapsed in any respect.

DISCUSSION

Two procedural problems which require clarification are (1) the optimal number of stimuli to be presented per session and (2) the optimal duration of stimulus presentations.

On theoretical grounds one would predict that the fewer stimuli presented at any one session, the more effective would be the result. Too many stimuli presented in close succession increase the risk of retroactive inhibition (interference) and there is abundant evidence to prove the superiority of distributed over massed practice.[7] Nevertheless, the therapist should not proceed too slowly as this might affect the patient's confidence in the treatment.* Regarding the duration of stimulus presentations, we have as a general rule used brief presentations (about 8 seconds) for the first few items in any hierarchy, increasing the time periods as a function of the patient's adaptation. No negative effects can be traced to this procedure, but we are not in a position to claim that this is the most efficient temporal arrangement. Further evidence on this specific point is required.

An extremely important problem raised by Wolpe is the transfer of 'consulting-room desensitization' to real-life situations. He quotes a dramatic example[4] of this transfer in a patient with a human-blood phobia who, very shortly after desensitization, was able to actively assist the victim of a serious road accident. Evidence is also presented by the case of a 20-year-old woman seen by one of us, on 5 occasions only, during a brief holiday in Johannesburg, who complained of long-standing acrophobia (among other things). After the second desensitization session, she was able to stand on a fifth-floor balcony without discomfort. Four months after her return home, she wrote to say that she had successfully undertaken a horse-riding excursion in very rugged mountain terrain. In addition to these two excerpts, the illustrative cases reported in this article also suggest that transfer is both positive and lasting.

The following are some additional impressions of the desensitization technique:

Our experience has been that the greatest benefit is derived when the therapist commences desensitization on the patient's most pressing current problem. An early desensitization success tends to speed up subsequent learning processes.

An observation not mentioned by Wolpe is the spontaneous recurrence of the anxiety symptom in some instances. In these cases, however, the spontaneous recurrence generally dissipates fairly rapidly and the patient returns to his improved condition with little further effort. These spontaneous

* In the present series 2-4 items were presented per session.

recoveries of former anxiety symptoms, while of brief and sporadic nature, must nevertheless be handled with extreme care because of the feelings of acute depression which they usually engender in the patient.

What conditions indicate systematic desensitization therapy? On theoretical and experimental grounds (see Gantt[11] and Jones[12] for example) it may be expected that phobic states, where concrete and definable stimuli produce the neurotic reaction, would be most amenable to this technique. In fact, wherever clinical symptomatology permits the ready construction of appropriate hierarchies, and where specific rather than 'free-floating' anxiety is present, systematic desensitization is strongly indicated.

SUMMARY

A detailed description of Wolpe's systematic desensitization psychotherapy is presented.

Five selected cases treated individually by one or other of the co-authors are described. The favourable results obtained indicate the value of these methods in the management of various types of neurotic disturbances.

Some clinical observations and additional suggestions which might assist in the development of the technique are noted.

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SILENT UTERINE RUPTURE RESULTING IN SECONDARY ABDOMINAL PREGNANCY

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Since its first mention in the Talmud, a monumental literature has accumulated concerning extra-uterine pregnancy. After the Rabbis of the Talmud first reported their observations of the 'child emerging from the abdominal side of the mother', and the Buddhist legend that the boy Buddha was 'born through the right side and armpit of his mother', the first accepted authentic account of the extra-uterine pregnancy was made by Albucasis in the 11th century. Later Riolan (1626) referred to several cases.

Credit for the first recorded operation for extra-uterine pregnancy must go to the New World. On 25 December 1796 John Bard, of New York, performed the laparotomy and the patient recovered.

Types of Ectopic Pregnancy. Ectopic is a pregnancy located outside the normal uterine cavity. Primary ectopic pregnancies comprise tubal, ovarian, abdominal and cervical. Tubal pregnancies occur in the ampulla, the isthmus, or the interstitial part of the tube. Abdominal pregnancies may be primary or secondary. In the primary the initial implantation of the fertilised ovum occurred in the abdominal cavity; the secondary are re-implanted after initial nidation at some other site. The cervical pregnancy is rarest, but implantation in the unfriendly mucosa of the cervix does occasionally occur.

In a diligent search of the literature no mention has been found of any case similar to the one about to be described.

CASE REPORT

L.T., a Native of about 29 years of age, was admitted to the Kimberley Maternity Hospital on 20 September 1953 complaining of vague abdominal pains.

Menstrual History: Menarche at about 13 years. Cycle regular 3/28 day type. No dysmenorrhoea.

Pregnancies 5; youngest now 2 years, oldest 14 years; 2 boys, 3 girls. No abortions, no operations.

Present History. Last menstrual period early in March 1953. During her pregnancy she experienced 'nothing different to her previous pregnancies'. She felt life after about 20 weeks. On 16 September 1953 she commenced feeling slight abdominal discomfort and was sent into hospital by her private doctor on 20 September 1953.

On examination the uterus appeared about the size of a 5 months' gestation; the cervix was hard and revealed several irregular lacerations. Abdominal palpation showed the presence of a live foetus lying transversely with the head to the mother's right flank, palpation being facilitated by an unusually thin abdominal wall. This was confirmed by X-ray, the radiologist describing the maturity as probably more than 6 months. She was kept in hospital for observation and it was hoped that the child would mature to viability.

Progress

On 13 October, strong labour pains began and persisted. Examination showed no cervical dilatation after 4 hours and no discharge, and at this stage the presence of an additional, and intra-uterine, foetus was excluded. The uterus was now distinctly larger than was first noted on admission. It was decided to operate.

During induction of anaesthesia the patient suddenly vomited and inspired some of the vomitus, becoming dyspnoeic, cyanosed and shocked, and rapidly developing signs of pulmonary oedema. The honorary anaesthetist to the hospital was called and expeditiously and successfully carried out a bronchoscopy, clearing the foreign matter from the bronchi by suction. She was taken back to the ward, where she rapidly recovered and the uterine contractions ceased.

On 19 October labour pains started again and operation was again decided upon. The honorary anaesthetist gave the anaesthetic, and warned us that the patient manifested signs of shock during the induction stage.

Through a sub-umbilical mid-line incision the abdomen was opened and an extraordinary picture was disclosed. A live, well developed foetus was found lying transversely with its head under the maternal liver, and enclosed in its membranes. Out of a porthole-like aperture in the wall of the uterus, about 4½

inches in diameter and encroaching onto the left cornu of the fundus of the uterus, emerged a cord of normal appearance and about 14 inches in length. When the cord was followed proximally through the orifice it was observed to enter a perfectly normal placenta firmly embedded in the myometrium adjoining the border of the orifice. Examination of the wall of the hiatus revealed that the outer edge was lined by peritoneum merging into smooth fibrous tissue covering the myometrium. A rapid survey revealed normal ovaries and tubes, and a uterus of about 5½ months' size.

My decision not to carry out hysterectomy nor to remove the placenta and attempt closure of the uterine window, was encouraged by an anxious anaesthetist. The cord was cut between ligatures and the 6½ lb. female child cried as it was lifted from the abdominal cavity. The membranes were freed from their attachments with no difficulty, and the abdomen closed without drainage.

The patient made a quite uneventful recovery from the operation. Unfortunately the baby, after doing well for the first few days collapsed on the 4th day and died.

On 10 November 1953 the mother, now well recovered, was taken back to the theatre and a hysterectomy was performed. During the operation about 2½ pints of blood-stained offensive-smelling fluid was sucked from the peritoneal cavity, in which,

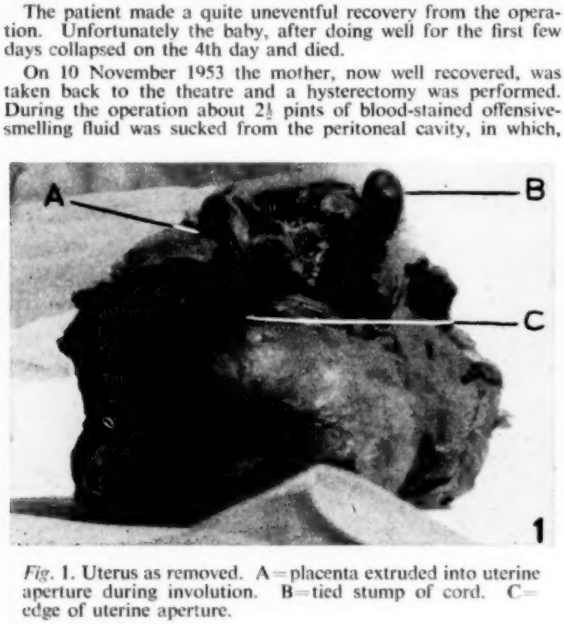


Fig. 1. Uterus as removed. A = placenta extruded into uterine aperture during involution. B = tied stump of cord. C = edge of uterine aperture.

however, no evidence of peritonitis could be discovered. Examination of the specimen (Fig. 1) showed only slight evidence of involution but the little that had taken place had squeezed the still firmly attached placenta into the uterine window where it bulged, giving a tumour-like appearance.

Close subsequent cross-examination failed to elicit from the patient any evidence that an accident, assault or other untoward event had taken place during her pregnancy which might account for the rupture of the uterus, which was apparently a symptomless episode, resulting in a secondary abdominal pregnancy.

The patient was discharged fit on 5 December 1953.

PATHOLOGIST'S REPORT

Macroscopic examination of this specimen of uterus and 'tumour' shows the presence of the uterine cavity and myometrium. Lateral to and above the uterus there is a large tumour-mass, 15 × 15 × 10 cm., attached to the myometrium.

Sections taken from the tumour show the presence of chorionic villi, which are fibrosed. The histological features are consistent with an extra-uterine pregnancy. There is no evidence of Fallopian tubes or ovarian tissue associated with the chorionic villi.

DISCUSSION

It must be accepted that originally fertilization and implantation of the ovum in the uterus took place as a normal event. It must also be accepted that rupture of the uterus occurred at some stage during pregnancy, with evacuation of the foetus through this false opening. At no time did the patient, a Bantu of average intelligence, complain to her doctor that anything unusual had happened during her pregnancy.

Examination of the healed uterine wound at the time of the first operation, of necessity a hasty scrutiny, cast little light on the stage of pregnancy at which rupture had taken place. One assumes that it occurred at the site of some weakness in the uterine muscle. On the other hand there was no history of external trauma, and the patient had never had a curettage or manual removal of an adherent placenta. Even if there had been evidence of any of these accidents, whence had come the stimulus for a rupture in this weakened section of the muscle? And why no haemorrhagic or other phenomena which might reasonably be expected to accompany such an event?

Are we left with the explanation that, perhaps following infection or embolus (neither very likely), a myometrial infarct developed which at an early stage of pregnancy became the seat of necrosis? One might suggest that in this way a plug of the uterine wall became dislodged, permitting the escape of the foetus.

My thanks are due to Dr. G. de V. de la Bat, Medical Superintendent of the Kimberley Hospital, for permission to publish this report, to Dr. B. Alexander, Honorary Anaesthetist, who carried out a life-saving bronchoscopy and both anaesthetics, to Dr. J. P. du Toit, at that time my House Surgeon, whose keen interest and energy contributed largely to this patient's eventual recovery and to whom I am indebted for the detailed notes available for reference, and to Mr. L. D. Pryde for the photographs of the specimen.

REFERENCE

Douglas, G. F., Douglas, G. C. and Douglas, G. F. Jr. (1951): J. Int. Coll. Surg., **15**, 28.

THE HISTORY OF MEDICINE IN SOUTH AFRICA

South Africa might be called the Daughter of Medicine. For was not the fight against scurvy the very reason for the establishment of the settlement at the Cape with its garden and hospital? Jan van Riebeeck was only the first among a surprisingly large number of leading South Africans whose activities were strongly influenced by their medical background, and the history of medicine in South Africa is much more than the story of the fight against illness and disease at the southern tip of the continent—it is linked intimately with the development of the whole country.

A medical man writing the history of medicine in South Africa would concentrate his research on the sometimes amazing medical achievements in a country where one would have expected a much more retarded development in medical practice than the rapid realization of medical ideas and the progressive achievements which actually took place. A historian on the other hand would emphasize the important role many medical men played under lawless conditions in consolidating a rather primitive society such as that of the first mining camps and the diamond

fields. Edmund H. Burrows, author of *The History of Medicine in South Africa to the End of the Nineteenth Century* (shortly to be published by A. A. Balkema under the auspices of the Medical Association of South Africa), is ideally equipped for his task. He is not only an authority on the history and genealogy of the Cape hinterlands, but is also a medical man—for this reason he succeeds in integrating the history of pioneering and colonization in all four provinces with the history of the development of medical practice, education and science.

The South African scene has at all times been rich in colourful personalities and Dr. Burrows has made full use of this advantage; his study is distinguished by more than 200 biographical sketches which are interspersed in the text. They represent a near-complete record of the individual contributions of the surgeons and doctors who established medicine from the Cape to the Limpopo and added their contributions to African progress. Among them are men like Dr. van der Kemp, William Stanger, Dr. H. Sauer, the remarkable Dr. Poortsman, L. S. Jameson and many others. These biographies give the book additional value. Besides being a comprehensive historical study, it will be consulted as a dictionary of medical biography. By sheer force of documentation (it contains nearly 1,850 references taken from 300 sources) the reader is convinced of the author's scholarly approach and reliability.

The story unfolds long before van Riebeeck's landing at Table Bay with the search for a sea-route to the East and the seventeenth century surgeons' fight against the shocking deathrate at sea. After the establishment of a half-way station at the Cape of Good Hope where sailors could be hospitalized, the development of the Colony is described, where a race of Cape burgers arose, among them the Berrangés, Flecks, le Sueurs, whose sons studied medicine in Europe and returned qualified to practise amongst their own people.

With the nineteenth century, medicine at the Cape progressed rapidly. The Batavian and British governments began to license practitioners and introduced the first public health measures such as vaccination. In Natal and in the hinterland medical practice developed independently from the nucleus of colonization at the Cape. The British settlers of 1820 brought with them 19 competent surgeons, but the great body of white farmers who crossed the Orange River in the Great Trek had to do without doctors and relied wholly upon their *boeremiddels* and the Hallische medicines of the Cape Town apothecaries. They were dependent upon the missionary 'doctor' such as Pellissier, Lautré, Lion-Cachet and Merensky, until a decisive change occurred with the arrival of that outstanding personality, Dr. Bernardus Poortman.

The author lays sufficient emphasis on the growth and influence of important hospitals in the Union and introduces the reader to the developments brought about by the great epidemics that raged in Cape Town. There arose a professional *esprit de corps* that led to the creation of an active medical society and the founding of the Cape Town Medical Gazette.

With the discovery of diamonds at Kimberley and of the Gold Reefs in the Transvaal, roistering multi-coloured mining communities sprang up and the fantastic life—and no less fantastic medical personalities mixed up in it—are portrayed here. Jameson's remarkable 'small-pox war' at Kimberley, Dr. Hans Sauer's vicissitudes in early Johannesburg, the jailed doctors of the Reform Committee, the whole exciting tale of mining, is mirrored in their exploits. In the Cape Colony, consolidation was now the key-note, and in the Eastern part—where Dr. W. G. Atherstone had used ether in an amputation as early as 1847—hospitals and lunatic asylums were built. Medical legislation guided by doctor M.P.s was passed and the 1890s saw the emergence of a Public Health Department and the appointment of the first full-time M.O.H. in Kimberley and Cape Town. Nursing came into its own and we find the beginnings of research and laboratory practice in the Cape.

In the final chapter the efforts of Sir Meiring Beck, Dr. Jane Waterson, the Fuller brothers and other pioneers of medical organization are traced.

A good number of maps and illustrations—among them historical photographs which are here published for the first time—add to the interest of the book. Judging from the loose proofs the standard of the typography is remarkably high, and the publisher's name is a guarantee that the production and binding of the book will be a credit to any library.

A prospectus is soon to be issued giving detailed information about the book, which will be made available on special terms to members of the Medical Association.

IN MEMORIAM

G. S. ANDREWS, M.B., B.CH. (RAND), D.M.R. (CAPE TOWN

Dr. Hugh Wormald, of Durban, writes: George Steven Andrews died at his home in Durban on 4 August 1957 after a short illness.

Dr. G. S. Andrews
Photo, Natal Mercury

He was born at Somerset Strand on 2 May 1909. On completion of his schooling in Johannesburg, he spent some years in the building and engineering trade before studying medicine at the University of the Witwatersrand, whence he graduated M.B., B.CH. in 1939.

After 6 years in the forces, during which time he served in the North with the 18th Field Ambulance, he decided to specialize in Radiology. While a postgraduate student of Radiology at the Cape he developed an intense interest in Radiotherapy, which always remained his first love. His early training and technical ability were to stand him in great stead in this particular sphere throughout his career.

I first became associated with 'Steve' in 1950, when we were colleagues in the Natal Provincial Radiological Service. From this association developed a firm friendship and a partnership which was unhappily to be terminated by his untimely death at the age of 48 years.

Steven Andrews was the most modest of men, a characteristic which manifested itself throughout his life. His sincere and conscientious application to the task in hand and his extreme honesty won him the highest respect from colleagues and patients alike. As a friend and partner there could be none better. He was most kind and considerate, and ever willing to lend a helping hand to those in need.

Steve Andrews held a most progressive outlook regarding his speciality and was never prepared to settle in a rut of routine procedure. He was a great believer in frank discussion with a view to improving cooperation and solving problems, and to this end he was prime mover in the foundation of the Natal Subgroup of the South African Radiological Society, which body he served most ably as Secretary in its first year. He was also Secretary of the Radiological Section of the forthcoming Medical Congress, and it was a great source of worry during his illness that he was unable to fulfil this commitment.

The death of Dr. Andrews is a great loss to the medical profession in general, and to Radiology in particular. At this time our heartfelt sympathy is extended to his wife, Peggy, his mother and his four children in their sudden and sad bereavement.

Dr. M. Denny, of Johannesburg, writes: With the passing of George Steven Andrews on 4 August 1957, the medical profession and, in particular, the speciality of Radiology lost a fine gentleman and great colleague.

I had the pleasure of knowing 'Steve' and his family for the last 10 years and, furthermore, had the privilege of working in his department at King Edward VIII Hospital, Durban. Those of us who were associated with him soon came to realize that here indeed was a gentleman endowed with that gracious gift so seldom found—modesty and a knowledge of his own limitations. With the passing of time it was equally clear that these limitations were few, and this particularly so in the field of Radiotherapy, his first love.

From him we learnt much of the basic principles of treatment. His humanitarian approach to those suffering from incurable malignant disease was welcome and refreshing. The high standard of Radiology at King Edward VIII Hospital, Durban, was largely due to the solid foundation laid by the efficient administration and progressive outlook of Steven Andrews.

One remembers well a farewell gathering of radiologists and clinicians just before he commenced private radiological practice. He said, 'I shall miss the great spirit of cooperation between clinicians and radiologists of this hospital', and, furthermore, expressed his fervent wish that this should continue unchanged. In the years that followed, he derived great pleasure from the fact that this spirit did indeed continue.

I am sure that his colleagues and many friends will join with me in expressing their deepest sympathy to his wife, Peggy, his mother, and his four children.

* * *

GYSBERT VAN REENEN MOSTERT, M.R.C.S., L.R.C.P.

Prof. L. J. te Groen van Pretoria skryf: Dr. Gysbert van Reenen Mostert het ek geken van die einde van die 1920er jare in die ou Pretoriase Algemene Hospitaal toe hy my huisdokter was. Hy het toe pas teruggekeer uit Engeland waar hy sy opleiding ontvang het.

Dr. Mostert

Dr. Mostert het hier in die regte gestudeer en na Londen gegaan met die bedoeling om dié studies daar te voltooi. In Londen het hy skielik van plan verander en as 'n mediese student by Guys-Hospitaal ingeskryf en daar sy graad behaal.

In sy skooldae was hy 'n uitstaande atleet en rugbyspeler.

Dr. Mostert was begaaf en bekwaam en was 'n goeie huisdokter. Hy was natuurlik ouer en ryper as die gemiddelde huisdokter. My vriendskap met hom dateer vanaf dié jare.

Hy het 'n vennoot van dr. Sthamer geword en later sy eie groot praktyk gehad, en hy is op die ere-personeel by die Algemene Hospitaal aangestel. Toe die Universiteit hier begin het was ek bly en gelukkig om hom as 'n deeltydse assistent in die Departement Obstetrie en Ginekologie te kon aanstel. Dit het hy uitstekend behartig totdat 'n beginnende katerak dit vir hom onmoontlik gemaak het om operasies te verrig. Hy moes ligter werk soek en vir 'n jaar was hy buite Pretoria werksaam. Sy katerak het toe dieselfde gebly en self daarna kon hy met behulp van atropine sy oë goed gebruik. Onder andere was dr. Mostert ook vir 3 jaar aangestel as Inspekteur van Interniste.

Twee jaar gelede het sy gesondheid agteruit gegaan en uiteindelik is hy geopereer vir 'n galblaas-ontsteking. Komplikasies het ingetree en hy was lank siek, maar het tog weer herstel. Vir die afgelope 2 jaar was hy by die Algemene Hospitaal as algemene mediese praktisyn werksaam.

Dr. Mostert was 'n ywerige werker vir die Mediese Vereniging. Hy was jare lank 'n lid van die Noord-Transvaalse Takraad, en nieteenstande sy swak gesondheid het hy gedurende 1955 as President van die Tak ageer. Dit was 'n bedrywige jaar vir die Noord-Transvaalse Tak, daar die 40ste Mediese Kongres van die Vereniging gedurende Oktober 1955 in Pretoria plaasgevind het.

Gedurende die afgelope twee weke het dr. Mostert se gesondheid skielik agteruit gegaan en op Saterdag, 17 Augustus is hy op die ouderdom van 59 jaar oorlede.

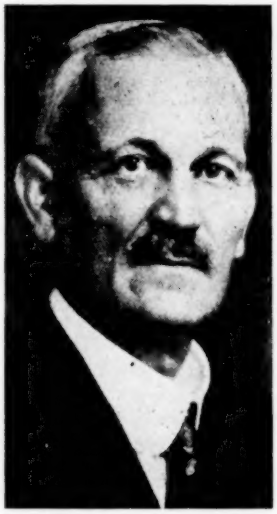
Dr. Mostert was 'n begaafde en bekwame dokter wat alles vir sy pasiënt en sy werk oor gehad het. Rustig, vriendelik van aard, goed gehumeurd met 'n groot sin vir humor—dit is te verstaan dat hy onder sy pasiënte en kollegas baie gewild was.

Hy het sy siektes en operasies moedig gedra. Die dood het vroeg gekom, maar miskien baie leed gespaar. Vir sy vrou en sy kinders is sy dood 'n groot verlies en my innige meegevoel gaan uit na hulle wat agterbly.

* * *

DR. R. H. WELSH

Dr. R. L. Girdwood, of Johannesburg, writes: It is my sad duty to express in our *Journal* a short appreciation of the grand work done in his lifetime by our colleague Dr. R. H. Welsh. I wish I were able to write about him as fully as the subject would justify.



Dr. Welsh

I met his family first in 1910 at Umtata, in the Transkei, when I did a short locum for him. I was tremendously impressed by the excellence of his work as District Surgeon and Superintendent of the local hospital. There being no specialists in those days all surgical work, medical and ear, nose and throat work, as well as gynaecology, was performed by Dr. Welsh. His surgery was of a high standard. I formed then the opinion, which I have never had occasion to alter, that he was a most wonderful practitioner. Both his European and Native patients almost worshipped him.

Robert Hepburn Welsh was born at Bedford, C.P. in 1868, and died at Witbank on 23 July 1957. His father was a Member of Parliament in the Cape Colony; his mother was Jessie Margaret Solomon, of Bedford, C.P. He married Harriet Ana Rose, first matron of Umtata Hospital.

He was educated at Dale College, Kingwilliamstown, and graduated at Aberdeen University in 1891. In 1892 he went to Umtata, succeeding Dr. Johnston. He was appointed District Surgeon of an area extending from the Bashee River to the Umzumvutu River. All his visiting had to be done on horseback. His post-mortems were performed in the face of active hostility on the part of the relatives of the deceased. He took an active part in forming the Sir Henry Elliott Hospital, of which he was medical superintendent from 1892 to 1929. He took a keen interest in the training of non-European nurses.

Dr. Welsh was a military medical officer in the Boer War and a Lieut.-Colonel in the 1st World War. He took an active part in medical affairs and was at one time a member of the Federal Council of the Medical Association of South Africa. He was much interested in the promotion of sport and education, and he was chairman of the Umtata School Board for many years.

He retired from practice for a short while in 1929. Later he joined his son-in-law in Witbank in a part-time capacity, only to shoulder the whole practice on the outbreak of World War II. Later, on full retirement, he was made an Emeritus Member of the South African Medical Association.

At the age of 86 Dr. Welsh flew to Britain and revisited his old University at Aberdeen, where he was welcomed as its oldest living graduate.

He leaves a son, a son-in-law and three grandsons, all members of the medical profession.

ABSTRACT : UITTREKSEL

PREVENTION OF TETANUS IN INJURED PERSONS*

PUBLISHED AT THE SUGGESTION OF THE WORKMEN'S COMPENSATION COMMISSIONER, PRETORIA

Whenever tetanus antitoxin is to be administered the following procedure should be strictly and carefully followed:

1. Keep a small sterile syringe and needle and a 1 : 1,000 solution of adrenaline ready for immediate emergency use. Keep the patient warm and under observation for at least 30 minutes after receiving antitoxin by any route.

2. If there is no previous history of serum administration and no history of allergy, give full dose of tetanus antitoxin, 1,500 international units subcutaneously or intramuscularly. If there are any doubts about previous injections or serum or allergic diathesis, proceed as in (3).

3. If the patient has had serum before but has no history of allergy give a 'trial dose' of 0·2 ml. of undiluted antitoxin subcutaneously. If no general symptoms develop within 30 minutes give a full dose of tetanus antitoxin 1,500 international units subcutaneously, or intramuscularly.

4. If history of allergy, give a trial dose of 0·2 ml. of 1 : 10 dilution of antitoxin subcutaneously. If no general reaction within 30 minutes give 0·2 ml. of undiluted antitoxin subcutaneously. If no general reaction within a further 30 minutes give full dose of tetanus antitoxin, 1,500 international units, subcutaneously or intramuscularly. The object of the trial dose is to determine any predisposition to general reaction and *not* to detect any local reaction, the latter having no practical significance.

TYPES OF REACTION WHICH MAY FOLLOW ADMINISTRATION OF ANTITOXIN AND THEIR TREATMENT

(a) *Anaphylactic Shock*

Onset within a few minutes, or, with less intensity, up to two hours. Dyspnoea, pallor, collapse: sometimes oedema or rash.

Treatment: Give 0·5 to 1 ml. of 1 : 1,000 solution of adrenaline intramuscularly. If recovery is not rapid repeat the injection in 15 to 20 minutes.

Give an antihistamine drug in full doses intramuscularly, such as Antistin 0·1 gm.—2 c.c.: specially useful if urticaria or oedema develops.

Keep the patient recumbent with the head low, and warm with blankets and hot-water bottles.

Recovery from anaphylactic shock is usually rapid after adrenaline. Very rarely, the return of blood pressure to normal is delayed for some hours; in these cases give a slow intravenous drip of diluted Noradrenaline, blood pressure reading being taken frequently.

(b) *Serum Sickness*

Common ('delayed') type after 7-12 days; 'accelerated' type after 3-4 days. Urticaria and other rashes, oedema of the tissues, joint pain and pyrexia.

Treatment: Give an antihistamine drug by mouth, such as Anthisan 2 tablets of 100 mg. each, for one to three days. In more serious cases antihistamine drug by injection; soothing lotions such as calamine to the skin.

In rare cases of severe serum sickness and when symptoms reappear in spite of antihistamine drugs, give Meticorten orally in doses of 5·0 mg. six-hourly for one or two days: discontinue the drug gradually over the next two days.

(c) *Local Reaction without Constitutional Disturbance*

This appears occasionally after 7-10 days, lasting two days. Localised erythema and urticaria; may be very irritating but never dangerous.

Treatment: Give an antihistamine drug orally; soothing lotion to the skin.

(d) *Thermal Reaction*

Sudden pyrexia and severe chill, mostly after intravenous injections. Rare.

Treatment: Give adrenaline as above. Keep patient warm.

*Parish, H. J., Laurent, L. J. M. and Moyinhan, N. H. (1957) Brit. Med. J., **1**, 639.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Charles Berman, M.D., M.R.C.P. is proceeding to New York, where he has been invited to join the Staff of the Sloan-Kettering Institute for Cancer Research as a Visiting Associate.

* * *

The Association of Physicians of South Africa. The annual meeting of this association will take place at 2.15 p.m. on Wednesday, 18 September in room 4, St. John's Hall, Durban. All members of Congress are kindly requested to attend.

* * *

Dr. Harold O. Hofmeyr, physician specialist, of 148 St. George's Street, Cape Town, and Mrs. Hofmeyr, have recently returned from a tour of the Continent, where Dr. Hofmeyr visited hospitals in Germany, England, France and Scandinavia.

* * *

Sir Charles Hastings and Charles Oliver Hawthorne Clinical Prizes, 1958. The Sir Charles Hastings Clinical Prize Competition is established by the British Medical Association for the promotion of systematic observation, research and record in general practice. The Competition has been extended by the addition of a second prize known as the Charles Oliver Hawthorne Clinical Prize. Enquiries relative to the prizes should be addressed to the Secretary, British Medical Association House, Tavistock Square, London, W.C.1.

Middlemore Prize 1958. The Council of the British Medical Association is prepared to consider an award of the Middlemore Prize (a cheque for £50 and a certificate) in the year 1958 to the author of the best essay on: 'Local Antibiotic Treatment in External Ocular Disease'. Notice of intention to enter for the competition should be made on the appropriate entry form, copies of which can be obtained from the Secretary, British Medical Association, B.M.A. House, Tavistock Square, London, W.C.1. Essays must reach the Secretary on or before 31 January, 1958.

* * *

Academic Year Day, University of Stellenbosch. The medical personnel of the Karl Bremer Hospital and the University of Stellenbosch are holding an Academic Year Day at the Civic Centre, Bellville, Cape, on 4 October 1957. Lectures will be given between 10 a.m. and 4 p.m.; medical films will be shown from 4 to 5 p.m. and scientific exhibits and pharmaceutical exhibits will be shown between 9 a.m. and 5 p.m. Luncheon will be served. All doctors are welcome. Further details of the programme are obtainable on application to Dr. A. J. Brink, Convener, Organizing Committee.

* * *

Die geneeskundige personeel van die Karl Bremer-hospitaal en die Universiteit van Stellenbosch kondig aan dat hulle 'n Akademiese Jaardag sal hou op 4 Oktober 1957 by die Burgersentrum, Bellville, Kaap. Voorlesings sal gehou word tussen 10 vm. en 4 nm.; mediese rolprente sal vertoon word tussen 4 en 5 nm.

en die farmaseutiese en wetenskaplike uitstallings word tussen 9 vm. en 5 nm. ten toon gestel. Middagete word bedien. Alle dokters is welkom. Verdere besonderhede kan op aanvraag van dr. A. J. Brink, Sameroeper, Organiserende Komitee, verkry word.

* * *

The Nathaniel Bishop Harman Prize 1958 *for Research in Hospital Practice.* The Council of the British Medical Association is prepared to consider the award of this prize in the year 1958. Its value is approximately £100. It will be awarded for the best report on original clinical research in a form suitable for publication. Candidates are required to complete an entry form, a copy of which can be obtained from the Secretary. Reports and entry forms must reach the Secretary, British Medical Association House, Tavistock Square, London W.C.1 not later than 31 January, 1958. No report or study that has previously been published in the Medical Press or elsewhere will be considered eligible for the prize.

* * *

The Southern African Cardiac Society. At a meeting of the Cape Province section of the Society held at Groote Schuur Hospital, Cape Town, on 28 August 1957, with Dr. Maurice Nellen, Chairman, and Dr. Louis Vogelpoel, Secretary, Dr. V. Schrire read a paper on the 'Diagnosis of Aortic Stenosis' by Dr. V. Schrire, Dr. H. Gordon and Mr. W. Phillips. Dr. Louis Vogelpoel also read a paper on 'A case of Ebstein's anomaly', and Dr. V. Schrire one on 'The Natural History of Pericarditis'. The annual general meeting of the society will be held at Durban at 2.15 p.m. on 20 September 1957. The Society now consists of the following sections: Cape Province, Natal, Transvaal and the Rhodesias.

* * *

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 23 August to 28 August 1957.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	–	1	–	–	1
Cape Province ..	–	2	–	–	2
Orange Free State ..	–	–	–	–	–
Natal	–	–	–	–	–
Totals	–	3	–	–	3

Plague, Smallpox, Typhus Fever: Nil.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Ribena. Ribena, a concentrated blackcurrent juice syrup, is now to become freely available for the first time in South Africa through the J. C. Eno organization, who supply the following information:

Clinical tests in hospitals in Britain have established its beneficial therapeutic effects in the treatment of: Hypovitaminosis-C (as in pregnancy and infancy); gastric and duodenal ulcer; blood dyscrasias, thrombopaenia, haemophilia and nutritional anaemias; acute febrile illnesses (e.g. pneumonia and rheumatic fever); toxic states (e.g. in diphtheria, scarlet fever and severe enteric infections); skin disorders; fractures and wounds; and post-operative conditions.

Ribena can be used to supply vitamin C to infants as early as the 4th week onwards;[1] and is particularly beneficial to premature babies.[2]

The value of Ribena in cases of peptic ulcer was demonstrated in experiments conducted by Adam Clark[3] wherein the results obtained were not duplicated by using synthetic vitamin C.[3]

The beneficial effect of vitamin C on the integrity of the skin has long been evidenced;[4,5] and cases of acne rosacea have been observed to clear up after a course of Ribena. Some cases of psoriasis have benefited.

Fixsen and Roscoe show the ascorbic-acid content of blackcurrent juice as 136-220 mg. per 100 g. as against 22-89 for orange juice. The average analysis of Ribena shows not less than 20 mg. of vitamin C per fluid ounce (55 mg. per 100 g.).

1. Ingalls, T. H. (1940): J. Pediat., **14,** 593.
2. Mackay, H. M. M. (1941): Brit. Med. J., **1,** 891.
3. Clark, A. (1950): April 7 and 14, Med. World.
4. Volpe (1937): Schweiz, Med. Wschr., **67,** 498.
5. Dainow, J. (1936): Brit. J. Derm., **48,** 167.

REVIEWS OF BOOKS : BOEKRESENSIES

MANUAL OF ANATOMY

A Manual of Human Anatomy. Vol. I. Thorax and Upper Limb. By J. T. Aitken, M.D., G. Causey, M.B., F.R.C.S., J. Joseph, M.D., M.R.C.O.G. and J. Z. Young, M.A., F.R.S. Pp. vii+162. 36 Figures. 14s. net plus 7d. Postage Abroad. Edinburgh & London: E. & S. Livingstone Ltd. 1956.

Contents: I. General Introduction to the Thorax. II. The Chest Wall. III. The Pleura. IV. The Lungs. V. The Mediastinum. VI. The Heart. VII. Other Mediastinal Structures. VIII. The Lymphatic Drainage of the Thorax. Suggestions for Osteology and Surface Anatomy Classes. IX. General Introduction to the Upper Limb. X. The Anterior Aspect of the Shoulder Girdle. XI. The Contents of the Axilla. XII. The Posterior Aspect of the Shoulder Girdle. XIII. The Shoulder Joint and its Stabilising Muscles. XIV. The Muscles Acting on the Elbow Joint. XV. The Forearm and the Back of the Hand. XVI. The Elbow and Radio-ulnar Joints. XVIII. The Palm of the Hand. XVIII. The Joints of the Wrist and Hand. XIX. The Cutaneous Nerve Supply. XX. The Lymphatic Drainage of the Upper Limb. Suggestions for Osteology and Surface Anatomy Classes. Index.

A Manual of Human Anatomy. Volume II. By J. T. Aitken, M.D., G. Causey, M.B., F.R.C.S., J. Joseph, M.D., M.R.C.O.G. and J. Z. Young, M.A., F.R.S. Pp. viii+180. 53 Figures. 16s. net+7d. Postage Abroad. Edinburgh & London: E. & S. Livingstone Ltd. 1956.

Contents: I. General Introduction. II. The Back of the Neck and the Trunk. III. The Spinal Cord and its Membranes. IV. The Face and Scalp. V. The Front and Sides of the Neck. VI. The Mouth and Tongue. VII. The Deep Dissection of the Neck. VIII. The Muscles of Mastication and the Mandibular Joint. IX. The Intracranial Region. X. The Orbital Cavity and Eyeball. XI. The Ear. XII. The Inferior Surface of the Base of the Skull. XIII. The Pharynx and Related Structures. XIV. The Larynx. XV. The Nasal Cavities. XVI. The Joints of the Cervical Vertebrae. XVII. The Lymphatic Drainage of the Head and Neck. XVIII. The Sensory Nerve Supply. Suggestions for Osteology and Surface Anatomy Classes. Index.

A Manual of Human Anatomy. Volume III. Abdomen and Pelvis. By J. T. Aitken, M.D., G. Causey, M.B., F.R.C.S., J. Joseph, M.D., M.R.C.O.G. and J. Z. Young, M.A., F.R.S. Pp viii +135. 29 Figures. 12s. 6d. net+6d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: I. General Introduction. II. The Anterior Abdominal Wall. III. The Abdominal Cavity. IV. The Abdominal Cavity (continued). V. The Abdominal Cavity (continued). VI. The Kidneys and Ureters. VII. The Diaphragm, Adrenal Glands and Abdominal Sympathetic Nervous System. VIII. The Posterior Abdominal Wall. IX. The Pelvic Viscera and Peritoneum. X. The Perineum. XI. The Perineum (continued). XII. The Prostate, Urethra and Vagina, Functional Aspects of the Pelvis and its Organs. XIII. The Muscles, Vessels and Nerves of the Pelvis. XIV. The Lymphatic Drainage of the Abdomen and Pelvis. Suggestions for Osteology and Surface Anatomy Classes. Index.

A Manual of Human Anatomy. Volume IV. Lower Limb. By J. T. Aitken, M.D., G. Causey, M.B., F.R.C.S., J. Joseph, M.D., M.R.C.O.G. and J. Z. Young, M.A., F.R.S. Pp. viii+117. 36 Figures. 12s. 6d. net+6d. Postage Abroad. Edinburgh & London: E. & S. Livingstone Ltd. 1956.

Contents: I. General Introduction to the Lower Limb. II. The Pelvic Girdle. III. The Lateral and Posterior Aspects of the Hip Joint. IV. The Anterior and Medial Aspects of the Hip Joint. V. The Hip Joint—Structure and Movements

VI. The Knee Joint. VII. The Ankle Joint. IX. The Foot. X. Posture and Walking. XI. The Cutaneous Nerve Supply of the Lower Limb. XII. The Venous and Lymphatic Drainage of the Lower Limb. Suggestions for Osteology and Surface Anatomy Classes. Index.

A Manual of Human Anatomy. Volume V. Central Nervous System. By J. T. Aitken, M.D., D. A. Sholl, B.Sc. and J. Z. Young, M.A., F.R.S. Pp. vii+140. 40 Figures. 12s. 6d. net + 6d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: I. General Introduction. II. Identification of Parts of the Brain and its Early Development. III. The Coverings of the Brain and the Spinal Cord. IV. The Blood Vessels and the Cranial Nerves. V. The Examination of the Nervous System. VI. The Spinal Cord. VII. The Medulla Oblongata. IX. The Pons. X. The Midbrain (Mesencephalon). XI. The Forebrain (1). XII. The Forebrain (2). XIII. The Histology of the Cerebral Cortex. Index.

The strenuous demands of a modern medical curriculum has unfortunately brought pressure to bear on the basic medical disciplines, anatomy and physiology, so that there is often a tendency to 'cut out' details in anatomy, to concentrate on what may only be required of the student in the clinical years and to increase the emphasis on function. The former two tendencies lead to many difficulties. Modern anatomical teaching should first endeavour to inspire the student to 'understand' human structure on the basis of 'pure' anatomy so that he may be able to apply his knowledge to function and disease and then, as a corollary, to train his mind in the principles of anatomy by using the logical approach of development (both embryological and evolutionary) structure, functional aspects and probable growth-phases or changes.

The authors have mainly followed this approach and have produced a reasonable dissection programme and a readable description of the parts dissected. The instructions are simple, the sketches large and clear. Medical schools have various methods of dissection and those that contemplate recommending these manuals to their students will have to organize a 'practice run' to determine whether the plan in the manuals suits their purposes (i.e. as regards the number of cadavers available, suitability of integration with the physiology course). The reviewer is favourably impressed not only by the authors' style and presentation but especially by the 'practicals' at the end of each volume where surface markings and osteology are planned for small tutorial classes. These would reduce the teaching load for these practicals would require only competent student demonstrators or junior lecturers.

The authors acknowledge that their course on neuro-anatomy (for University College, London) may be in excess of what may be considered necessary elsewhere for the second medical examination; but why all the emphasis should be placed on the central nervous system and so little on the autonomic nervous system is difficult to understand in the light of the aims set out in the preface of each volume.

The reviewer feels obliged to take the authors to task on a few points: In each preface they state that 'the study of each part can thus be undertaken with some knowledge of the functional implications of the anatomy and not as a mere exercise of memory. Much detail has been omitted'. Very few anatomists can honestly aver that their experience of anatomy has not come about by much exercising of the memory. Functional aspects are of assistance, but the bases of *understanding* in anatomy are thorough dissection, clear visual memorizing, correlation of features and a solid foundation of embryology. Furthermore, although it is 'popular' to add occasional functional aspects of structures in modern text-books, anatomy as a discipline cannot *at the same time* embody physiology—the two should be integrated in the lecture theatre. The amount of functional aspects of, say, the duodenum, presented in these manuals may link it with the pancreas and gall-bladder, but cannot claim to frame its position in the student's mind as regards its relationship to peritoneum, the right kidney or the transpyloric plane. However, no one can deny the importance of providing the opportunity for the student to appreciate the mechanical and kinesiological aspects of the locomotor apparatus so that he may grasp the essentials of myology, arthrology and osteology.

This argument leads on to the second sentence quoted above. How much should be omitted? This depends on the number of hours available and not on the commonest diseases encountered in the clinical years. The reviewer considers that the authors have almost struck a reasonable balance, but he feels that if repetitions (and not of major features at that) had been avoided and additional facts inserted in their place, many sections would have been of more value to the student. A second-year anatomy course can be made 'attractive' by sacrificing some detail but this should not be done on the altar of over-applied anatomy. For example, in volume I, much of the general introduction is repeated in the following chapter and the repetition is not particularly of 'essential' points. Instead of repeating fig. 1 (vol. 1) as fig. 15 (60 pages on), it may have been more advisable, say, to provide a diagram of the structure of a vertebra to illustrate the text. The illustration of the ossification centres of the os innominatum may have given way for one showing the attachments to the bone, especially in the light of such a loose phrase as 'This is the tensor fasciae latae, which arises from the iliac crest in front' (Vol. IV, page 6), which by its vagueness will disturb the student more than ease matters for him. No mention is made of the communications between the superficial and deep veins of the lower limb.

A few of the weak features are the over-simplification of the lymph drainage and the scanty descriptions of the coronary arteries, the mammary gland, the internal ear, the peritoneum, and the costo-vertebral joints, while some of the surface anatomy instructions are cumbersome, e.g. of the heart. There are minor errors throughout the manuals, but when the reviewer considers the books in their entirety, these criticisms do not weigh heavily against the distinct value of the series, and he has no hesitation in recommending them to South African anatomy departments (especially those with small staffs) for a 'trial run' with a view to permanent adoption. However, he advises that these manuals should be considered purely on their merits as laboratory guides. They in no way replace the standard text-books. They may 'cover' the course, but the majority of South African students would need to refer to their text-books to supplement their knowledge to meet the requirements of the second-year examiners.

The manuals are well printed and wire-backed, each having a differently coloured cardboard cover, which unfortunately will wear badly.

R.S.

CORRESPONDENCE : BRIEWERUBRIEK

PENICILLIN REACTIONS IN ALLERGIC SUBJECTS

To the Editor: In a recent Editorial[1] of this *Journal*, I was surprised to read that the use of penicillin in asthmatic subjects was recommended.

Welch *et al.*[2] record a total of 84 anaphylactoid reactions to penicillin. Thirty-one per cent had a history of bronchial asthma, hay fever or other allergies and 14% had previous evidence of sensitivity to penicillin, 28% proved fatal. Although these reactions are rare they are sufficiently alarming and serious to be significant. Acute anaphylactoid reactions attributable to penicillin are more numerous than indicated by published reports as severe anaphylactoid reactions may occur in a physician's office or elsewhere without being recorded.

In view of the serious nature of these reactions the Food and Drug Administration issued a memorandum to members of the antibiotic industry as a warning on 20 May 1953 stating: 'The administration of penicillin in rare instances may cause acute anaphylaxis. The reaction appears to occur more frequently in patients with bronchial asthma or other allergies and those who have previously demonstrated a sensitivity to penicillin . . .'

Welch and his co-workers in this same survey also disclosed no anaphylactoid reactions attributable to chloramphenicol, chlortetracycline, oxytetracycline, bacitracin, polymyxin or neomycin.

Much has been written about the prophylaxis of these acute and sometimes fatal reactions to penicillin therapy. Careful history-taking, fundamental to the practice of medicine, is a *sine qua non* in the avoidance of these reactions. In view of the above, a personal or even family history of allergy, e.g. asthma, hay fever, or childhood eczema is a contra-indication to the use of penicillin and according to Eales[3] its administration in these cases may result in catastrophe. Most of the fatalities with penicillin have been attributed to profound circulatory failure or to asphyxia due to intense bronchospasm[4]—a further reason to avoid its use in a patient who already has bronchospasm.

To prevent accidents in patients with allergic diathesis, hypersensitivity tests are imperative and desensitization is necessary, if penicillin treatment is indispensable. Even desensitization is not a safeguard against a severe or fatal reaction. It is best to treat an allergic patient with a broad-spectrum antibiotic and not with penicillin.[5]

Asthma and hay fever by their very nature, are recurrent maladies and respiratory infection a common concomitant and consequently antibiotic therapy is frequently indicated. This is yet another reason against the use of penicillin in these subjects, since the incidence of allergic reactions in patients after previous penicillin medication is 25·4% as opposed to 5·4% in patients without previous penicillin medication.- Even oral administration is not free from reactions. Sensitization and severe reactions may occur after oral administration.[2,7,8]

True, the use of penicillin is recommended together with cortisone which may protect allergic subjects against untoward reactions. Mathews *et al.*[9] have shown that the simultaneous administration of an antihistamine has been without significant protective value. Pick and Patterson[10] and Winton and Nora[11] by recording their fatal cases illustrated the danger of relying on simultaneous administration of either adrenalin or cortisone in sensitized individuals.

Although penicillin, in my opinion, is contra-indicated in allergic subjects, e.g. in asthma and hay fever, its use is entirely justifiable and can be used with great advantage in the non-sensitized and allergic-free patient, if there is a definite indication. In the allergic individual (with a past, present or family history of allergy) where an antibiotic is indicated, a broad-spectrum one is justifiable. According to Ioannides and McC. Murdoch[12] tetracyclines should be reserved for long-term treatment of more chronic advanced cases of bronchial infection and the short-term treatment of acute episodes should be treated with chloramphenicol. The potential toxicity of chloramphenicol has been overstressed. The above antibiotics are more effective and above all, are safer in allergic subjects.

Philip Lanzkowsky
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1. Editorial (1957): S. Afr. Med. J., **31,** 867.
2. Welch, H., Lewis, C. N., Kerlan, I. and Putnam, L. E. (1953): Antibiot. and Chemother., **9,** 891.
3. Eales, L. (1955): S. Afr. Med. J., **29,** 970.
4. Lewis, G. W. (1957): Brit. Med. J., **1,** 1153.
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6. Peck (1948): as quoted by 'Physician'.[5]
7. Bell, R. C. (1956): Lancet, **2,** 439.
8. Calvert, R. J. and Smith, E. (1955): Brit. Med. J., **2,** 302.
9. Mathews, K. P., Hemphill, F. M., Sheldon, J. M., Lovell, R. G. and Forsythe, W. E. (1955): J. Allergy, **26,** 78.
10. Pick, F. J. and Patterson, J. F. (1953): Brit. Med. J., **2,** 605.
11. Winton, S. S. and Nora, E. D. (1955): Amer. J. Med., **18,** 66.
12. Ioannides, A. H. and Murdoch, J. McC. (1957): Brit. Med. J., **1,** 1157.

TORN CONGENITAL DISCOID MENISCUS OF KNEE

To the Editor: The article by Dr. E. C. Greubel-Lee[1] in the current number of the *Journal* I found most interesting and stimulating.

During the past year I have seen two patients with congenital lateral meniscal lesions which I consider are thought-provoking in the light of the conclusions reached by the author.

The first patient was an adult male who presented with a 'locked knee' following a typical internal derangement. At operation a lateral discoid semilunar cartilage was found with a bucket-handle tear. The major portion of the meniscus was displaced centrally as shown diagrammatically in Fig. 1. In addition the centrally displaced portion of the meniscus was cleft horizontally to form a deep pocket.

The second patient was also an adult male. The presenting symptoms in this instance suggested a systic lateral semilunar cartilage. At operation a 'reversed' semi-discoid lateral semilunar cartilage was found. I have tried to illustrate this in Fig. 2, but regret that I did not have a camera available to photograph the

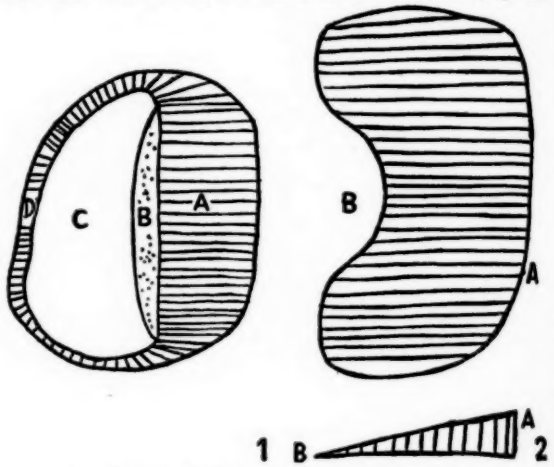

Fig. 1. (*a*) Central displaced portion of meniscus. (*b*) Opening into cleft. (*c*) Tear. (*d*) Peripheral attached portion of meniscus.

Fig. 2. (*a*) Central attached margin. (*b*) Peripheral, free, notched margin. Cross section below.

unusual state of affairs. In this instance the meniscus was firmly fixed centrally and had a peripheral free notched margin. In addition the central fixed attachment was the thickest part of the cartilage, which thinned out towards the periphery. On removal the deep surface of the cartilage (i.e. the tibial surface) was found to be very corrugated.

In my experience most of the discoid cartilages removed in adults have shown one of the features found present in the first case. The second case, which I feel sure is not unique, is strong presumptive evidence of the more usually accepted theory of meniscal development. I postulate that, for some reason or other, instead of the usual central absorption and thinning of the middle of the cartilage, this occurred in the peripheral margin in this instance.

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1. Greubel-Lee, E. C. (1957): S. Afr. Med. J., **31,** 852 (24 August).

BOOK REVIEW INITIALS

To the Editor: I wonder whether the time has not come for the abolition of the rule of anonymity which is presumed to cloak your book reviewers?

The rule may be satisfactory if universally applied, but when some reviewers who are fortunate (?) enough to hail from north of the Tweed, are able, to all intents and purposes, to circumvent it by a judicious use of 'Mac this and Mac that', it tends to become farcical.

MacMinde
Queenstown
2 September 1957